POTATO DISEASES

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POTATO DISEASES

By Muriel J. O'Brien and Avery E. Rich

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PREFACE

This handbook is addressed to scientists, professional and technical workers, to plant protection officials, students, and other persons interested in plant diseases in general and potato diseases in particular, and to commercial growers. It is a compilation of the major diseases and related problems of potato and recommends measures for preventing or reducing losses from these problems. The handbook also contains a glossary of scientific and technical terms commonly used in potato-disease literature on pages 74 through 77, and tables of helpful measurements on page 78. This publication supersedes U.S. Department of Agriculture Farmers' Bulletin 1881, "Potato Diseases and Their Control," issued October 1941, and revised February 1948.

Potato diseases are caused by fungi, bacteria, viruses, mycoplasmas, a viroid, nematode infestations, and by abiotic, or noninfectious, entities. Insects such as leafhopper and psyllids cause injuries to the potato that are often confused with diseases caused by infectious organisms. All disease problems derive from the interaction of many factors. Among these factors are: (1) The plant's vigor, or its lack; (2) the environmental conditions during plant growth; (3) the conditions at harvesttime; (4) proper storage of the tubers; and (5) the presence of disease organisms or insects, or the occurrence of physiological disturbances.

Preventive or control measures have been developed for most potato-disease problems. These control measures include, in some instances, the use of chemical fungicides, bactericides, and insecticides. All chemicals mentioned in this handbook should be applied in accordance with directions on the manufacturer's label as registered under the Federal Insecticide, Fungicide, and Rodenticide Act. Because chemical uses and specific dosages vary in different parts of the United States and are geared to the type farming done in the area, consult your State agencies for local recommendations.

Trade names are used in this publication solely for the purpose of providing specific information. Mention of a trade name does not constitute a guarantee or warranty of the product by the U.S. Department of Agriculture or an endorsement by the Department over other products not mentioned.

For data on pesticide dosages, tolerances, and other derivative information, consult the following U.S. Department of Agriculture publications:

Chemical Control of Plant-Parasitic Nematodes (Agriculture Handbook 286)

Controlling Potato Insects (Farmer's Bulletin No. 2168)

The Golden Nematode Handbook (Agriculture Handbook 353)

The authors wish to acknowledge that Raymon E. Webb, Chief, Vegetable Laboratory, Plant Genetics & Germplasm Institute, Agricultural Research Service, contributed some figures used in this publication.

COMMON, TRADE, AND CHEMICAL NAMES OF SELECTED PESTICIDES

Common name	*Trade name*	*Chemical name*
bordeaux mixture	—	mixture of copper sulfate with suspension of calcium hydroxide.
captafol	Difolatan®	cis-N-[(1,1,2,2,-tetrachloroethyl)thio]-4-cyclohexene-1,2-dicarboximide.
chloropicrin	Dowfume® MC–33 Larvacide®	trichloronitromethane.
chlorothalonil	Bravo W–75® Daconil 2787® DAC 2787® Termil®	2,4,5,6,-tetrachloroisophthalonitrile.
DD	—	1,3-dichloropropene and 1,2-dichloropropane and related chlorinated C_3 hydrocarbons.
1,3–D	Telone®	1,3-dichloropropene.
DD-MENCS	Vorlex®	80% DD + 20% methylisothyocyanate.
disulfoton	BAY 19639 Dithio-Systox® Disyston® Solvirex	*0,0*-diethyl *S*-[2-(ethylthio)ethyl]phosphorodithioate.
EDB	Bromofume® Dorlone® Dowfume® W–85 Garden Fume® Pestmaster® EDB–W85 Soil Fume Caps® Soilbrom–85® Soilfume 83®	ethylene dibromide or 1,2-dibromoethane.
formaldehyde	—	formaldehyde.
maneb	MEB Dithane® M–22 Manzate®	manganous ethylenebis [dithiocarbamate].
neutral (fixed) copper	—	copper oxychloride sulfate and oxide and other forms of low soluble copper fungicides.
PCNB	quintozene Terrachlor® Terraclor® Terraclor Super X®	pentachloronitrobenzene.
sulfur	—	sulfur.

Common name	*Trade name*	*Chemical name*
SMDC	Vapam® VPM Trimaton® Chem-Vape® Mycoban® metam methamsodium	sodium *N*-methyldithiocarbamate.

PRECAUTIONS

Pesticides used improperly can be injurious to man, animals, and plants. Follow the directions and heed all precautions on the labels.

Store pesticides in original containers under lock and key—out of the reach of children and animals—and away from food and feed.

Apply pesticides so that they do not endanger humans, livestock, crops, beneficial insects, fish, and wildlife. Do not apply pesticides when there is danger of drift, when honey bees or other pollinating insects are visiting plants, or in ways that may contaminate water or leave illegal residues.

Avoid prolonged inhalation of pesticide sprays or dusts; wear protective clothing and equipment if specified on the container.

If your hands become contaminated with a pesticide, do not eat or drink until you have washed. In case a pesticide is swallowed or gets in the eyes, follow the first aid treatment given on the label, and get prompt medical attention. If a pesticide is spilled on your skin or clothing, remove clothing immediately and wash skin thoroughly.

Do not clean spray equipment or dump excess spray material near ponds, streams, or wells. Because it is difficult to remove all traces of herbicides from equipment, do not use the same equipment for insecticides or fungicides that you use for herbicides.

Dispose of empty pesticide containers promptly. Have them buried at a sanitary landfill dump, or crush and bury them in a level, isolated place.

NOTE: Some States have restrictions on the use of certain pesticides. Check your State and local regulations.

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POTATO DISEASES

By MURIEL J. O'BRIEN, *plant pathologist,* and AVERY E. RICH,[1] *visiting plant pathologist, Plant Genetics and Germplasm Institute, Northeastern Region, Agricultural Research Service, U.S. Department of Agriculture, Beltsville, Md.*

INTRODUCTION

The potato, *Solanum tuberosum* L., is grown throughout the United States. It is one of the most important and most widely distributed of our food crops. Although acreages in potato cultivation have declined in this country, the yield per acre has increased since the 1940's due, in large measure, to the efficient control of insects and diseases. Total annual potato production fluctuates between 200 million and 300 million hundredweight.[2]

Plant diseases, certain pests and injuries, and unfavorable environmental conditions cause potato losses. The estimated loss from potato pathogens is 19 percent, or 38 to 57 hundredweight annually. Important fungus diseases that contribute to this loss are late blight, 4 percent; Verticillium wilt, 2.7 percent; common scab, 1.5 percent; early blight, 1 percent; and Rhizoctonia canker, 1 percent. Blackleg and ring rot are the most serious bacterial diseases, each causing 0.5 percent loss. Virus-disease losses include leafroll, 3 percent; and, for the mosaics, 1 percent each for rugose mosaic, mild mosaic, and latent mosaic.

Losses occur from attacks by nematodes and from the plant injuries produced by the feeding of insects. [Descriptions of insects that attack potato and recommended methods for their control are available in other U.S. Department of Agriculture publications.] This Handbook lists only two insect injuries on potato, injuries that could possibly be mistaken for other diseases. Finally, losses result from the occurrence of abiotic diseases. These diseases are produced by unfavorable environmental conditions and are not transmitted by insects or other disease-causing agents.

The prevention of disease occurrence and of disease spread is of primary importance to successful potato production. Disease occurrence is usually termed "endemic," "epidemic," or "sporadic." Endemic diseases are those that occur naturally; they are endemic, or indigenous, to the locality. The organisms responsible for endemic diseases can survive for long periods, often under adverse conditions, in the soil or on a weed host. Under the proper environmental conditions, endemic diseases develop and spread. Epidemic diseases are those that occur widely and usually cause considerable damage. Sporadic diseases occur rarely and at irregular intervals in a particular locality or over a wide geographic area.

Diseases develop in the field, in storage, in transit, in the market, and in the home. Disease loss has been reduced, however, by the use of resistant varieties. Planting potato varieties that are resistant to one or more diseases is one effective means of combating potato-disease losses. Many varieties used in the United States have been derived from breeding work done in the National Breeding Program, which is conducted cooperatively by State agricultural experiment stations and the U.S. Department of Agriculture. Some of the States have also independently developed varieties that are suitable for their own specific growing conditions and economic needs. Good cultural practices also help to prevent disease losses. Rotation is essential. Potato should be grown in a planned crop rotation to insure production of the largest yields per acre.

[1] Dr. Rich was an ARS visiting pathologist, February–September, 1967; he is now associate dean, College of Life Sciences and Agriculture, University of New Hampshire, Durham.

[2] U.S. DEPARTMENT OF AGRICULTURE. AGRICULTURAL STATISTICS. 627 pp. Washington. 1970.

Chemical pesticides, such as fungicides, bactericides, and insecticides, are also used to control potato diseases. These pesticides are applied to the soil, used to treat seed, and are used as foliar sprays or dusts. A list of the chemical names of pesticides mentioned in this handbook begins on page iii.

The use of clean seed is also important in successful potato production. Diseases can be prevented or limited in their occurrence if certified, disease-free seed is planted in clean soil in areas free from the weeds that carry disease-producing organisms and harbor insects.

USE CLEAN SEED IN CLEAN SOIL. USE CLEAN EQUIPMENT WHEN HANDLING ALL PRODUCTION PROCEDURES.

To recognize and identify the various diseases and disorders of the potato and to use the proper measures for their control, the investigator should be able to diagnose a disease or related problem from the symptoms found on various parts of the potato plant. The gross morphology of a growing plant often indicates the presence of a disease or disorder. Examination of the entire plant, of leaves, stems, tubers, sprouts, stolons, and roots for lesions or discolorations can often be made macroscopically. At other times, microscopic examination of affected tissues and the structure of the causal organisms may be necessary. Often, the attacking organism must be grown on artificial media before it can be identified.

Many disease problems are easily recognized, but other diseases may be difficult to identify properly, particularly when more than one disease is present or a disease complex exists. Where there is a question as to the identity of a disease and its control, consult your local county agricultural agent or send specimens to your State agricultural college or university.

To assist in the preliminary identification or diagnosis of potato disease problems, a modified key has been prepared that describes the more salient disease symptoms found on potato plants, stems, leaves, leaflets, tubers, stolons, sprouts, and roots. In each of these plant-tissue categories, the abnormalities in color, texture, shape, size, and internal discolorations or derangements are treated. For the most part, the problems in each category are listed in the ascending order of symptom severity. Complete descriptions of the diseases and disorders, with recommended control measures, follow this key and the alphabetized, synoptic table (table 1) of the diseases treated in this handbook.

MODIFIED KEY TO POTATO DISEASES AND DISORDERS

PLANTS

A. PLANTS PRESENT

a. Plants *abnormal* in *color*

Plants yellowish green	Leafroll, p. 49

b. Plants *abnormal* in *shape*

Plants pyramidal in growth habit	Psyllid yellows, p. 60
Plants with rosetted appearance, terminal growth malformed	Haywire, p. 46
Plants with tufted appearance	Golden nematode, p. 57
Plants with numerous, weakened, spindly stems	Witches' broom. p. 56
Plants with branching, stalky shoots arising from aboveground axillary buds	Psyllid yellows, p. 60
Plants with leaves subtended at 45° angle	Spindle tuber viroid, unmottled curly dwarf virus, p. 53.
Plants oriented clockwise when viewed from apex	Do.

c. Plants *abnormal* in *size*

Plants compact	Phyllid yellows, p. 60
Plants occasionally dwarfed	Corky ring spot, p. 45
Plants slightly dwarfed	Golden nematode, p. 57
Do	Haywire, p. 46
Do	Interveinal mosaic, paracrinkle, p. 46.
Do	Latent mosaic, potato virus X, p. 47.
Do	Leafroll, p. 49
Do	Witches' broom, p. 56

AA. PLANTS ABSENT

Plants in uneven stand	*Rhizoctonia solani*, p. 29
Plants missing	Fertilizer burn, p. 63
Plants dying prematurely	Root-knot nematodes, p. 59
Plants dead	Lightning injury, p. 67

STEMS

A. STEMS NORMAL

a. Stems *normal* with *outward lesions*

Lesions *throughout stem*

Irregular necrotic streaks on stems and petioles	Stem streak necrosis, p. 69

Lesions *above* or *near* the *ground*

Light-colored, water-soaked lesions on lower stem; white mycelium present later	Sclerotinia white mold, [*Whetzelinia*], (stalk break), p. 33.
Sparse, whitish growth on stems above soil line	*Rhizoctonia solani*, p. 29
White, gray, or purplish-gray feltlike mycelium near ground surface	*Thanatephorus cucumeris* (perfect stage of *R. solani*), p. 29.
Lesions as dark, soft, shallow rot on lower stems	Charcoal rot, p. 21
Lesions dark, sometimes slimy, inky-black and extending upward	Blackleg, p. 39

Lesions *below ground*

Dark-brown lesions on underground stems	*Rhizoctonia solani*, p. 29
Dark, brown, cortical rot of underground stem	Skin spot, p. 34
Lesions sunken, discolored; water-soaked white, fanlike mycelial growth on underground stem surface; mycelial mats with sclerotia resembling mustard seed	Sclerotium rot (southern blight), p. 33.

aa. Stems *normal* but with *inward lesions*

Rust-colored specks in pith of upper part of stem	Yellow dwarf, p. 56
Brown flecks in pith of lower part of stem	Purple-top wilt, p. 51
Inner tissue stained brown, often from base of plant well into top	Verticillium wilt, p. 35
Do	Wilt (*Fusarium oxysporum*), p. 22.
Inner tissue stained brown from base of plant into top *and* extending to upper leaflets	Wilt (*Fusarium solani* var. *eumartii*), p. 22.
Vascular bundles brown	Brown rot (southern bacterial wilt), p. 40.
Do	Purple-top wilt, p. 51
Stem interior filled with mycelium and black sclerotia	Sclerotinia white mold, [*Whetzelinia*], (stalk break), p. 33.

AA. STEMS ABNORMAL

a. Stems *abnormal* in *color*

Stems and vines darker than normal	Fertilizer burn, p. 63

b. Stems *abnormal* in *shape*

Petioles and stems sometimes swollen at nodes	Haywire, p. 46
Stems spindling and numerous	Witches' broom, p. 56
Aerial tubers developing along entire stem	Psyllid yellows, p. 60
Aerial tubers formed in leaf axils	Haywire, p. 46
Green or reddish aerial tubers formed in leaf axils	*Rhizoctonia solani*, p. 29

—Continued

LEAVES

A. LEAVES NORMAL IN GROWTH HABIT

a. Leaves with *surface markings*

Silvering or glazing on lower leaf surfaces	Air pollution damage (PAN), p. 61.
Dark-brown stippling on upper leaf surface	Air pollution damage (ozone), p. 61.
Irregular, dark-brown to black spots on leaves	Late blight, p. 25
Leaves with brown, nonglossy dead spots	Early blight, p. 21
Leaves with concentric rings in a target-board pattern	Early blight, p. 21
Leaves with compact, white mycelial growth; mycelial growth *nonglistening*	Powdery mildew, p. 28
Leaves with aerial, white mycelial growth; mycelial growth *glistening;* sporangia sometimes present on underside of leaves	Late blight, p. 25

aa. Leaves with *subsurface markings*

Leaves with large, irregular, yellow to cream-colored spots	Calico, p. 45
Leaves with necrotic spots	Latent mosaic, potato virus X, potato mottle virus, p. 47.
Leaves with small, numerous, mottled areas	Rugose mosaic, potato virus Y, p. 52.
Leaves with diffuse mottling	Interveinal mosaic, paracrinkle, p. 46.
Leaves mottled with yellowish or pale-green spots interspersed in normal green of foliage	Mild mosaic, potato virus A, p. 50.
Do	Latent mosaic, potato virus X, p. 47.
Leaves with dead areas between veins and along leaf margins	Bacterial ring rot, p. 43
Veins of lower leaves showing dead, pencillike streaks or areas on inside of leaf surface	Rugose mosaic, potato virus Y, p. 52.
Leaves darker green than those of healthy plants	Spindle tuber viroid, p. 53

b. Leaves with *abnormal color*

Leaves sometimes yellowish	Verticillium wilt, p. 35
Do	Wilt (*Fusarium oxysporum*), p. 22.
Leaves yellow	Blackleg, p. 39
Leaves yellowish green	Yellow dwarf, p. 56
Leaves yellowish, often conspicuously so	Bacterial ring rot, p. 43
Leaves reddish yellow, sometimes purple, leaf margins yellowed	Psyllid yellows, p. 60

c. Leaves with *abnormal texture*

Upper leaves soft	*Rhizoctonia solani*, p. 29
Leaves usually soft or leathery	Purple-top wilt, p. 51
Leaves somewhat velvety	Witches' broom, p. 56
Leaves slimy	Bacterial ring rot, p. 43
Leaf surface slightly crinkled	Mild mosaic, potato virus A, p. 50.
Leaves slightly wrinkled and ruffled	Interveinal mosaic, paracrinkle, p. 46.
Leaves leathery and papery to touch, leaves sometimes brittle; lower leaves rolled	Leafroll, p. 49
Leaf margins rolling; dead tissues of leaves brittle	Hopperburn, p. 60
Leaves brittle	Rugose mosaic, potato virus Y (current-season infection), p. 52.

d. Leaves *wilting* or *dying*

Leaves wilting	Brown rot (southern bacterial wilt), p. 40.
Leaves rolling and wilted	Blackleg, p. 39
Do	Verticillium wilt, p. 35
Do	Wilt (*Fusarium oxysporum*), p. 22.
Leaves rolled at margins, then wilting	Bacterial ring rot, p. 43
Outer leaves dying back	Golden nematode, p. 57
Shoots often dying from tip downward	Yellow dwarf, p. 56
Leaves dying, drooping, or dropping off entirely	Rugose mosaic, potato virus Y (current-season infection), p. 52.
Leaves drying up and becoming tan	Sunscald, p. 71
Leaves and vine growth killed	Lightning injury, p. 67

AA. LEAVES ABNORMAL IN GROWTH HABIT

a. Leaves *with surface markings*

Leaves malformed and mottled with irregular, necrotic spotting	Corky ring spot, p. 45

aa. Leaves *without surface markings*

Leaves small, rounded	Witches' broom, p. 56
Leaves rolling	Purple-top wilt, p. 51
Upper leaves rolling	*Rhizoctonia solani*, p. 29
Leaves set at an acute angle with main stem	Spindle tuber viroid, p. 53
Abnormal number of axillary yellowish shoots formed, swollen at base	Purple-top wilt, p. 51
Aerial tubers formed in leaf axils	Purple-top wilt, p. 51

A. LEAFLETS ABNORMAL

a. Leaflets *abnormal* in *color*

Leaflets wilted, light green at first	Sunscald, p. 71
Leaflets with yellow mottling and blotching	Calico, p. 45
Leaflets (upper) with bronzing and slight yellowing	Wilt (*Fusarium solani* var. *eumartii*), p. 22.
Leaflets purple at tips and margins	Haywire, p. 46
Leaflets gradually dying and with blackening at tips and margins	Hopperburn, p. 60
Leaflets with tips and margins yellowing, then gradually dying with browning or blackening of leaflets	Tipburn, p. 71

b. Leaflets *abnormal in shape*

Leaflets pointed	Haywire, p. 46
Leaflets erect	Do.
Leaflets stiff	Do.
Leaflets rolled	Do.
Leaflets rolling upward	Leafroll, p. 49
Upward rolling of basal part of smaller leaflets on young plants	Psyllid yellows, p. 60
Leaflets rolling upward with pronounced rolling at their bases	Purple-top wilt, p. 51
Leaflets with wavy margins	Mild mosaic, potato virus A, p. 50.

c. Leaflets *abnormal* in *texture*

Leaflets of lower leaves papery in texture	Leafroll, p. 49
Leaflets rugose	Haywire, p. 46
Leaflets with surface and margins uneven or crinkled	Rugose mosaic, potato virus Y, p. 52.
Leaflets with ruffled margins	Mild mosaic, potato virus A, p. 50.

TUBERS

FIELD

Seed piece firm; marble-sized tubers growing directly from eye	Sprout tubers (secondary tuber formation), p. 69.
Seed piece completely rotted	Blackleg, p. 39
Tubers small, abundant	Witches' broom, p. 56
Tubers small, abnormally abundant, unmarketable	Psyllid yellows, p. 60
Tubers lacking, or few, and borne close to stem	Haywire, p. 46
Tubers killed	Lightning injury, p. 67

HARVESTED

A. TUBERS NORMAL IN SHAPE

a. Tubers with *surface defects*

Tubers with *discoloration* on *surface*

Skin reddish brown, often cracked	Bacterial ring rot, p. 43
Tubers with metallic gray or brown surfaces	Leak, p. 27
Tubers with light- to dark-green pigment on skin	Sunburn (greening), p. 70
Tubers with small, grayish or discolored spots	Lesion nematode, p. 58
Tubers with dull, dark-brown, water-soaked appearance	Pink rot, p. 28
Tubers with gray surface, darkening with age	Phoma tuber rot, p. 28
Tubers with moist brown or black areas on surface	Blackheart, p. 62
Tubers with light to dark-brown spots on surface that often coalesce and become silvery and glassy when wet	Silver scurf, p. 34
Tubers with wet surfaces when thawed	Frost, or freezing necrosis, p. 64.

b. Tubers with *slight proliferations* on *surface*

Tubers with slightly sunken spots	Sclerotium rot (southern blight), p. 33.
Tubers with pimples that become black depressions	Lesion nematode, p. 58
Tubers with sunken areas of firm, brown rot	Dry rot (*Fusarium* spp.), p. 23.
Tubers with scablike openings on skin; tissue around openings raised and corky	Enlarged lenticels, p. 63
Tubers with papery skin, skin tearing and appearing as a ragged buttonhole	Phoma tuber rot, p. 28
Tubers with numerous, small, russetted areas covering entire tuber surface	Scab, common (surface type), p. 30.
Tubers with roughened appearance, with brownish, rounded, irregularly shaped, corky rough spots	Scab, common (pitted type), p. 30.

Tubers with small, dark pustules on tuber surface; pustules dark brown outside, olive brown inside	Skin spot, p. 34
Tubers with circular, blisterlike pustules that break open in stellate fashion; pustules with brown, powdery spore mass	Scab, powdery, p. 32
Small, slightly raised pustulelike elevations on tuber surface; pearl-white centers beneath pustules	Root-knot nematodes, p. 59
Tubers with dark, shallow, sunken areas; margin of diseased area raised, adjoining skin puckered, irregularly shaped, with raised borders of gunmetal hue surrounding sunken areas	Early blight, p. 21
Tubers with brown or black sclerotia on surface	Rhizoctonia canker, p. 29
Tubers with bacterial ooze and soil sticking to eyes	Brown rot (southern bacterial wilt), p. 40.
bb. Tubers with *distinct proliferations* on *surface*	
Tubers showing distinct "eyebrowing" at eyes	Spindle tuber viroid, p. 53
Tubers with proliferations, or knobs, at one or more eyes	Second growth (knobby tubers), p. 68.
Tubers with warty, cauliflowerlike outgrowths at eyes of tubers	Wart, p. 37
Tubers with galls or swellings, large at times; tubers with knotty appearance	Root-knot nematodes, p. 59
Tubers with bluish or white protuberances	Dry rot (*Fusarium* spp.), p. 23
aa. Tubers with *internal defects*	
Tubers with *discoloration beneath surface*	
Tubers with greenish yellow or deep-yellow color in flesh beneath skin	Sunburn (greening), p. 70
Tubers with light-gray to coal-black lesions beneath skin	Internal black spot, p. 66
Tubers with *discoloration* in *vascular elements*	
Tubers with golden-yellow to dark-brown discoloration in vascular tissue; discoloration pronounced in vascular-ring tissues at either end of tuber and between ring and tuber surface	Heat and drought necrosis, p. 64.
Tubers with shallow discoloration in vascular ring at stem end *only;* discoloration limited to about one-half inch from stem end	Stem-end browning, p. 54
Tubers with internal discoloration in and around vascular ring	Frost, or freezing necrosis (ring type), p. 64.
Tubers with dark-brown discoloration in vascular ring	Brown rot (southern bacterial wilt), p. 40.
Do	Wilt (*Fusarium solani* var. *eumartii*), p. 22.
Browning or blackening of vascular-ring tissues	Verticillium wilt, p. 35
Do	Wilt (*Fusarium oxysporum*), p. 22.

Network of brown to dark-brown dead fibers throughout stem end of tubers and extending *beyond* one-half inch from stem end	Phloem (net) necrosis [Leaf-roll], p. 49.
Discoloration of xylem and phloem elements darker than in phloem (net) necrosis	Stem-end browning, p. 54
Tubers with internal discoloration as fine strands radiating from vascular tissue to pith and outer tissues	Frost, or freezing necrosis (net type), p. 64.
Tubers with brown discoloration of xylem ring, discoloration extending *throughout* length of tuber	Xylem ring discoloration, p. 72
Rot in vascular ring gray, creamy yellow, or light to red brown	Bacterial ring rot, p. 43
Sticky, milk-white exudate coming from brown vascular strands	Brown rot (southern bacterial wilt), p. 40.
Vascular rot crumbly	Bacterial ring rot, p. 43
Tubers with *discoloration throughout tuber flesh*	
Tubers with brown spots in flesh	Wilt (*Fusarium solani* var. *eumartii*), p. 22.
Tubers with small necrotic areas scattered throughout the flesh	Yellow dwarf, p. 56
Tubers with irregular, dry, brown spots throughout the flesh	Internal brown spot, p. 67
Tubers with brown flesh to considerable depth or throughout the entire tuber	Sunscald, p. 71
Tubers with irregular, opaque gray or blue to sooty black patches anywhere in tuber	Frost, or freezing necrosis (blotchy type), p. 64.
Tubers with caramel-colored, sugary rot sometimes throughout entire tuber	Late blight, p. 25
Rot in tubers white to cream-colored, soft, watery	Bacterial soft rot, p. 39
Rot in tubers at first white, buttery in consistency, rot later turning black and slimy	Blackleg. p. 39
Tubers with inky-black tissue when cut (late stages)	Leak, p. 27
Grayish or brownish granular tissue decay (late symptoms)	Lesion nematode, p. 58
Tubers with soft, watery, and granular tissues	Leak, p. 27
Tubers with watery, rubbery, dirty-white tissue that turns pink on exposure to air	Pink rot, p. 28
Tubers watery in late stages	Sunscald, p. 71
Entire tuber slimy	Sclerotium rot (southern blight), p. 33.
Tubers with *discoloration* in *center* of *tuber*	
Internal gray to purplish black discoloration in heart of tuber	Blackheart, p. 62
Tubers with *discoloration* and *cavity* in *center* of *tuber*	
Irregularly shaped cavity in center; tissue browned	Hollow heart, p. 66

AA. TUBERS ABNORMAL IN SHAPE

a. Tubers *with surface defects*

Tubers irregular, rusty-brown concentric rings on tuber surface	Corky ring spot, p. 45
Tubers sometimes cracked	Yellow dwarf, p. 56
Tubers rough, round, with deep growth cracks	Unmottled curly dwarf, p. 53
Tubers withered, dry, wrinkled, sunken and tough at stem end	Jelly-end rot, p. 24
Tubers discolored, brown or black at stem end with a sharp line between diseased and healthy tissues	Jelly-end rot, p. 24

aa. Tubers *without surface defects*

Tubers small, misshapen	Yellow dwarf, p. 56
Tubers irregular	Corky ring spot, p. 45
Tubers oval, elongated, blocklike, and cylindrical in shape; pointed at stem end	Spindle tuber viroid, p. 53
Tubers with pointed ends	Second growth (knobby tubers), p. 68.

b. Tubers with *internal defects*

Irregular, rusty-brown lesions in tuber flesh; discolored flesh corky in texture	Corky ring spot, p. 45

STOLONS

A. STOLONS ABNORMAL

a. Stolons *abnormal* in *color*

Brown discoloration present in vascular bundles	Brown rot (southern bacterial wilt), p. 40.
Do	Purple-top wilt, p. 51

SPROUTS

A. TUBER SPROUTS ABNORMAL

Tuber sprouts abnormally slender and feeble	Leafroll, p. 49
Do	Spindling sprout (hair sprout), p. 68.
Do	Witches' broom, p. 56

ROOTS

A. ROOTS ABNORMAL

Roots with small galls or knots on surface	Root-knot nematodes, p. 59
Roots excessively branched	Do.

TABLE 1.—*Common diseases of potatoes*

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
Air pollution damage	--	Ozone and PAN.	Leaves	--	--	Avoid polluted areas; use varieties that resist this type of injury.
Bacterial soft rot	*Erwinia carotovora.*	--	Tubers	Soil and wash water.	Soil	Handle tubers carefully to avoid wounding; harvest in dry weather, dry tubers after washing; store at cool temperatures and in dry atmosphere; avoid frost injury.
Bacterial ring rot (see Ring rot).						
Blackheart	--	Insufficient oxygen; storing tubers at high temperatures.	Tubers	--	--	Avoid high-temperature storage; provide adequate ventilation; remove tubers promptly from soil surface when dug during hot weather.
Blackleg	*Erwinia phytophthora.*	--	Vines and tubers.	Soil, seed, and seedcorn maggot.	Soil and seed.	Use disease-free seed; use whole or suberized cut seed; provide good sanitation; destroy cull piles; and practice crop rotation.
Botrytis gray mold	*Botrytis cinerea*	--	Leaves, petioles, and tubers.	Soil, air, water.	Soil; plant debris.	Spray with a fungicide; provide proper storage conditions.
Brown rot	*Pseudomonas solanacearum.*	--	Vines and tubers.	Tubers and soil.	Tubers and soil.	Use disease-free seed; practice crop rotation; acidify soil with sulfur.
Calico	Alfalfa mosaic virus.	--	Vines and tubers.	Sap, aphids, diseased tubers.	Seed	Use disease-free seed; rogue disease plants; destroy volunteer alfalfa plants; control insects.
Charcoal rot	*Macrophomina phaseolina.*	--	Tubers	Soil	Soil	Grow in cool soil; use cool storage.
Corky ring spot	Cause unknown; (?) tobacco rattle virus.	--	Vines and tubers.	Soil, seed, nematodes.	Soil and seed.	No control methods known but use disease-free seed; avoid infested soil.

TABLE 1.—*Common diseases of potatoes*—Continued

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
Early blight	*Alternaria solani.*	--	Leaves and tubers.	Airborne spores.	Soil; plant debris.	Practice crop rotation; provide proper nutrition and water; control other diseases and insects; spray or dust with a fungicide.
Enlarged lenticels	--	Leaving tubers in wet soil after digging; storing freshly dug tubers in very moist atmosphere.	Tubers	--	--	Remove tubers promptly from field at harvesttime and store properly; supply proper irrigation; level the land to eliminate wet spots.
Feather and scald	--	Rough handling of immature tubers.	Tubers	--	--	Harvest tubers after they have matured; handle tubers carefully; do not expose tubers to wind or sun for extended periods.
Fertilizer burn	--	High nitrogen concentrations near seed pieces and roots.	Roots and seed pieces.	--	--	Follow proper fertilizer placement.
Frost or freezing necrosis; low temperature injury.	--	Ice formation in tuber tissues; extended low-temperature storage.	Tubers	--	--	Harvest and store crop before ground freezes; store potatoes at proper temperature.
Fusarium wilt, tuber rots, and seed-piece decay.	*Fusarium* spp.	--	Vines and tubers.	Soil and seed.	Soil and seed.	Avoid infested fields; avoid wounding tubers; use healthy seed; treat seed; practice crop rotation; follow good soil management.
Gangrene (see Phoma tuber rot).						

TABLE 1.—*Common diseases of potatoes*—Continued

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
Golden nematode	*Heterodera rostochiensis.*	--	Vines, roots, and tubers.	Soil and tubers.	Seed and soil.	Practice good sanitation; fumigate soil; heed quarantines; follow good field practices; plant resistant varieties; use nematode-free seed.
Green dwarf disease (see Haywire).						
Hair sprout (see Spindling sprout).						
Haywire	Sugar beet curly-top virus.	--	Vines and tubers.	Seed and beet leafhopper.	Seed and weeds.	Use disease-free seed; rogue affected plants; control insects and weeds.
Heat and drought necrosis.	--	Leaving tubers in hot soils after vines begin to die.	Tubers	--	--	Keep soil moist, cool, and shaded; harvest tubers promptly if soil is light and weather hot.
Hollow heart	--	Uneven growing conditions; conditions that favor rapid growth.	Tubers	--	--	Space plants closely; apply adequate fertilizer and irrigation; kill vines.
Hopperburn	--	Feeding of potato hopper.	Leaves	--	--	Control potato leafhopper with an insecticide; control leafspot diseases; use cultural practices that will ensure adequate soil moisture.
Internal black spot	--	Bruising; harvesting tubers before maturity; effects of top killing the vines; dry soil.	Tubers	--	--	Handle tubers carefully; provide proper storage; plant resistant varieties.
Internal brown spot	--	Hot, dry weather; lack of soil moisture.	Tubers	--	--	Provide proper irrigation; use favorable cultural practices; plant resistant varieties.

TABLE 1.—*Common diseases of potatoes*—Continued

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
Interveinal mosaic	Potato viruses X, S, and M.	--	Vines and tubers.	Sap, seed, aphids.	Seed	Select disease-free seed; control aphids with insecticides; practice good sanitation.
Jelly-end rot	*Fusarium solani f. radicicola.*	--	Tubers	Soil	Soil	None; maintain uniform and adequate soil moisture during growing season.
Late blight	*Phytophthora infestans.*	--	Leaves, vines, and tubers.	Airborne spores, water, seed.	Seed, cull piles, volunteer potato plants.	Plant only healthy seed potatoes; eliminate cull piles; spray or dust with a fungicide; kill vines 10 days before harvest; plant resistant varieties.
Latent mosaic	Potato virus X	--	Vines and tubers.	Sap and seed	Seed	Practice sanitation; use disease-free seed.
Leafroll	Leafroll virus	--	Vines and tubers.	Aphids and seed.	Seed	Plant resistant varieties; use disease-free seed; isolate the seed plots; rogue infected plants; control aphids.
Leak	*Pythium* spp.	--	Tubers	Soil	Soil	Harvest tubers when mature and during cool weather; use cool storage; handle tubers carefully to avoid bruises.
Lesion nematodes	*Pratylenchus* spp. and other spp.	--	Roots and tubers.	Soil	Seed and soil.	Fumigate soil; provide good sanitation; practice crop rotation; use nematode-free seed.
Lightning injury	--	Lightning	All parts	--	--	None.
Low-temperature injury (see Frost or freezing necrosis).						
Mild mosaic	Potato virus A	--	Vines and tubers.	Seed and aphids.	Seed	Use disease-free seed; control insects; rogue diseased plants; plant resistant varieties.

TABLE 1.—*Common diseases of potatoes*—Continued

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
Phoma tuber rot	*Phoma exigua* var. *foveata.*	--	Tubers (on stems in Europe).	Soil	Soil	Handle tubers carefully to avoid wounds; store tubers at 65° F (18 to 19° C) for 10 days to promote rapid healing of wounds.
Pink rot	*Phytophthora erythroseptica.*	--	Vines and tubers.	Soil	Soil	Avoid excessive irrigation; remove infected tubers; avoid wounding tubers; use cool, dry storage.
Potato rot nematode	*Ditylenchus destructor.*	--	Tubers	Soil and seed	Soil and seed.	Use nematode-free seed; practice strict sanitation; practice crop rotation; fumigate soil; field dry the tubers.
Powdery mildew	*Erysiphe cichoracearum.*	--	Petioles, stems, and (?) leaves.	Airborne spores.	(?) Weeds	If severe, use sulfur sprays.
Psyllid yellows	--	Feeding of potato psyllids.	Vines and tubers.	--	--	Control psyllids with systemic insecticides.
Purple-top wilt	Aster yellows "virus," (?) Mycoplasma.	--	Vines and tubers.	Leafhoppers and seed.	Weeds and seed.	Control leafhoppers; use disease-free seed; rogue diseased plants; control weeds.
Rhizoctonia canker	*Rhizoctonia solani* [*Thanatephorus cucumeris*].	--	Stems and tubers.	Soil and seed	Soil and seed.	Use disease-free or disinfected seed; practice crop rotation; employ shallow covering; treat soil.
Ring rot	*Corynebacterium sepedonicum.*	--	Vines and tubers.	Tubers and equipment.	Tubers and storage.	Use disease-free seed; use whole seed; maintain strict sanitation; practice crop rotation; plant resistant varieties.
Root-knot nematodes	*Meloidogyne* spp.	--	Roots and tubers.	Soil	Soil and seed.	Fumigate soil; avoid use of nematode-infested land; use (Con.)

TABLE 1.—*Common diseases of potatoes*—Continued

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
						nematode-free seed; practice crop rotation; follow good sanitation procedures; summer fallow the land to kill weeds.
Rugose mosaic	Potato virus Y	--	Vines and tubers.	Aphids, sap and seed.	Seed	Use disease-free seed; control insects; rogue diseased plants; use resistant varieties.
Scab, common	*Streptomyces scabies.*	--	Tubers	Soil and seed	Soil and seed.	Plant resistant varieties; plant scab-free or disinfected seed potatoes in scab-free soil; maintain soil pH at 5 to 5.2; choose proper fertilizers and cover crops; avoid overliming; practice long rotations.
Scab, powdery	*Spongospora subterranea.*	--	Tubers	Soil and seed	Soil and seed.	Avoid infested soil and infected seed; rotate crops; plant seed in warm soils.
Sclerotinia white mold (Stalk break).	*Sclerotinia sclerotiorum* (=*Whetzelinia sclerotiorum*).	--	Vines and stems.	Soil, airborne spores, and water.	Soil; plant debris.	Follow good sanitation practices; use crop rotation; flood land.
Sclerotium rot	*Sclerotium rolfsii.*	--	Vines and tubers.	Soil and plant parts; crates and bags.	Soil and other vegetable crops.	Avoid harvesting and bagging in wet weather; dry tubers before bagging.
Second growth	--	Uneven growth conditions.	Tubers	--	--	Provide uniform growing conditions and proper water supply; plant resistant varieties.
Secondary tuber formation (see Sprout tubers).						

TABLE 1.—*Common diseases of potatoes*—Continued

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
Silver scurf	*Spondylocladium atrovirens.*	--	Tubers	Soil and seed	Soil and seed.	Use disease-free seed; practice crop rotation; harvest tubers as soon as mature; store tubers in cool, dry place.
Skin spot	*Oospora pustulans.*	--	Tubers	Soil and seed	Soil and seed.	Use disease-free seed; practice crop rotation.
Southern bacterial wilt (see Brown rot).						
Spindle tuber	Potato spindle tuber viroid.	--	Vines and tubers.	Sap, seed, and insects.	Seed	Use disease-free seed; rogue infected plants; follow good sanitation practices; complete hilling and cultivation when plants are small; control insects.
Spindling sprout (hair sprout).	--	Tuber weakness.	Tubers	--	--	Presprout seed.
Sprout tubers	--	Excessive concentration of cell sap in tubers.	Tubers and plants.	--	--	Place tubers in cool, dark storage; practice late planting.
Stalk break (see Sclerotinia white mold).						
Stem streak necrosis	--	Manganese toxicity.	Stems and petioles.	--	--	Lime the soil; apply fertilizer; plant resistant varieties.
Stem-end browning	Cause unknown; (?) virus.	--	Tubers	Seed	Seed	Plant resistant varieties; use proper storage temperature.
Sunburn	--	Sun and artificial light.	Tubers	--	--	Hill plants properly in field; store tubers in complete darkness; market tubers in light-proof containers; rotate potato tubers on display in market.
Sunscald	--	Sun and wind.	Leaves and tubers.	--	--	None, but prevent exposure of tubers to (Con.)

TABLE 1.—*Common diseases of potatoes*—Continued

Disease name or disorder	Causal agent	Contributing factor	Part of plant affected	Mode of transmission	Seasonal carryover	Control
						sun for extended periods of time.
Tipburn	--	Hot, dry weather; feeding of leafhoppers.	Leaves	--	--	Conserve soil moisture; control insects.
Unmottled curly dwarf (see Spindle tuber).						
Verticillium wilt	*Verticillium* spp.	--	Vines and tubers.	Soil and tubers.	Soil and seed.	Avoid planting infected seed; avoid use of infested soil; practice crop rotation; control weeds; plant resistant varieties.
Wart	*Synchytrium endobioticum.*	--	Tubers and vines.	Soil and seed	Soil and seed.	Eradicate diseased plants; heed quarantines; avoid use of infested fields; use certified disease-free seed; practice sanitation and crop rotation; plant resistant varieties.
Witches' broom	Virus suspected; (?) mycoplasma.	--	Vines and tubers.	Seed and leafhoppers.	Seed	Use disease-free seed; rogue diseased plants.
Xylem ring discoloration.	--	Vinekillers	Tubers	--	--	Select slow-acting vinekillers; use mechanical beaters.
Yellow dwarf	Potato yellow dwarf virus.	--	Vines and tubers.	Seed and leafhoppers.	Seed, clover, and leafhoppers.	Use disease-free seed; rogue diseased plants; control insects.

FUNGUS DISEASES

Fungi are complex micro-organisms that often cause plant diseases. Fungi are usually characterized as possessing a threadlike vegetative growth (mycelium). They reproduce by means of structures that are called spores. These can be asexual or sexual forms and are variously called chlamydospores, conidiospores, sporangiospores, swarmspores or zoospores; ascospores, basidiospores, and oospores.

Some fungi that attack potato live in the soil; others overwinter in or on potato tubers and in plant debris. They enter the plant or tubers

through wounds or through natural openings, such as lenticels, or by direct penetration of the epidermis. Direct penetration is effected by a germ tube or peg. Once fungi gain entrance to a leaf or tuber, their mycelial strands either ramify directly into the cell, or grow between the cells.

Fungi are disseminated by wind, rain, soil, insects, drainage water, infested equipment, and by the infected tubers. The environmental conditions surrounding the plant have a direct effect on disease incidence and development. Some fungus diseases are favored by cool, moist weather; other diseases develop under warm, relatively dry conditions. When the proper interaction of environment and disease incitant occurs, an epidemic usually results.

The fungi that attack potato are found in all classes of fungi: Phytomyxinae, Phycomycetes, Ascomycetes, Basidiomycetes, and the Fungi Imperfecti. Except for the Phytomyxinae, a class that comprises the single order Plasmodiophorales, the fungi that attack potato can be found in various orders and families within each class. Table 1 lists the common fungus potato diseases, the specific fungus that causes each disease, and the recommended control.

Botrytis Gray Mold

Causal agent: Botrytis cinerea Pers. ex Fr.

Gray mold is a common fungus disease that has a wide host range. It is usually considered to be saprophytic or only weakly parasitic. Occasionally, it attacks potato plants and tubers.

The fungus grows as a gray or brownish, nonglistening mold on infected leaves and petioles usually under cool, humid conditions. Gray mold is sometimes confused with late blight infection, but *Phytophthora infestans* (Mont.) D By., the causal agent for late blight, produces a white, glistening fungus growth in contrast to the gray or brown, dusty fungus growth produced by *Botrytis*. Gray mold frequently attacks the older, lower leaves that are weakened by shading and old age.

Control

Botrytis gray mold can be controlled with a fungicidal spray. Store potatoes at a warm temperature (18° to 20° C) to hasten periderm formation. After storage for 2 weeks at the warm temperature, place potatoes in a cold storage (5° to 10°).

Charcoal Rot

Causal agent: Macrophomina phaseolina (Tassi) G. Goidanich
=*M. phaseoli* (Maubl.) Ashby

Charcoal rot is a common disease of many vegetables in the Mediterranean area, in the Southern United States, and in central California, as well as in other warm-region countries of the world. The disease is usually of minor importance on potato, but when the weather is warm and the soil wet, maturing plants may be attacked.

The charcoal-rot organism is a soil-inhabiting fungus and on some plants it is a root-inhabiting organism. It thrives under warm, moist conditions. In potato, the seedpiece is probably infected first. The fungus then moves into the roots, the lower stem area, and the stolons, then progressing to the tuber. In the tuber it incites a decay. A soft, dark-colored, shallow rot develops on lower stem area that is frequently followed by secondary organisms.

This organism is highly variable in spore form, and pycnidial and nonpycnidial strains exist. The fungus persists in the soil by producing sclerotia which are the dormant, resistant structures of this fungus. Soil fungistasis and exudations from the roots of the host have an effect on germination of these sclerotia. Some evidence exists that the severity of the disease (at least for jute (*Corchorus capsularis* L.)) is affected by a deficiency of potash in the soil.

Control

In the South, plant early-maturing varieties. Harvest tubers before hot weather occurs and store them in a cool place.

Early Blight

Causal agent: Alternaria solani (Ell. & G. Martin) Sor.

Early blight is a leaf-spot disease that most commonly attacks potato stems and leaves. On stems this disease causes a brown-black necrosis, and on leaves the symptoms of this dis-

ease appear as round, oval or angular, dark brown to black, nonglossy necrotic spots. These spots usually have concentric rings that produce a bull's eye, or target-board effect (fig. 1). The spots tend to be limited by the larger leaf veins. These small, scattered, dark spots are usually produced on the lower, senescent leaves, which often become yellow.

Early blight sometimes attacks the tuber. On tubers the lesions are small, sunken, round or irregular in shape, with slightly raised margins. The skin around these margins is slightly puckered (fig. 2). The affected tissue also develops a brown, corky dry rot. Lesions are usually 1 to 2 centimeters (cm) in diameter and less than 6 millimeters (mm) deep (fig. 3). Tuber lesions may afford an entrance for saprophytes, or secondary soil-inhabiting fungi, which then complete the rotting of the tuber.

Alternaria solani overwinters in plant refuse in the soil. It is usually considered to be a weak parasite. Plants that lack vigor are predisposed to attack by this fungus. Improper fertilizer levels (such as lack of nitrogen), and virus infection, and insect damage are examples of factors predisposing the plant to infection. High temperatures and high humidity favor development of early blight, but rain is not necessary for the development of the disease. The fungus usually attacks the leaves 2 or 3 weeks before harvest. When the spots are numerous, they kill the leaves and consequently reduce the yield of potatoes.

Figure 1.—Early blight lesions on a potato leaf.

Figure 2.—Early blight lesions on the tuber.

Control

To control early blight: (1) Practice crop rotation; (2) maintain healthy, vigorous foliage by providing proper nutrition and water; (3) adequately control other diseases and insects; and (4) spray or dust with an effective fungicide. The new organic fungicides are more effective and less phytotoxic than bordeaux mixture and other copper fungicides. Difolatan, chlorothalonil, and maneb are effective control measures. (See also recommendations for control of late blight.)

Fusarium: Wilt, Tuber Rots, and Seed-Piece Decay

Causal agent: Various species of *Fusarium.*

Fungus species in the genus *Fusarium* cause a variety of potato troubles. *Fusarium* spp. cause wilt, tuber rots, dry rot of tubers in storage, and seed-piece decay. *Fusarium* spp. are soilborne. They are an extremely complex group of fungi, difficult to identify with accuracy as to species, form, and physiologic race. About 20 species have been identified on potatoes.

Wilt.—Fusarium wilt is caused by *F. oxysporum* Schlecht. emend. Snyd. & Hans. and *F. solani* (Mart.) Appel & Wr. var. *eumartii* (Carpenter) Wr. *F. oxysporum* is more widespread but it is less pathogenic than *F. solani. F. solani* causes severe losses in the irrigated valleys of the Great Plains States. Fusarium wilt is also the most common disease of potatoes in the West, especially on those grown in irrigated lands that have warm soils.

In the wilt caused by *F. oxysporum* and *F. solani* var. *eumartii,* there is a yellowing of the

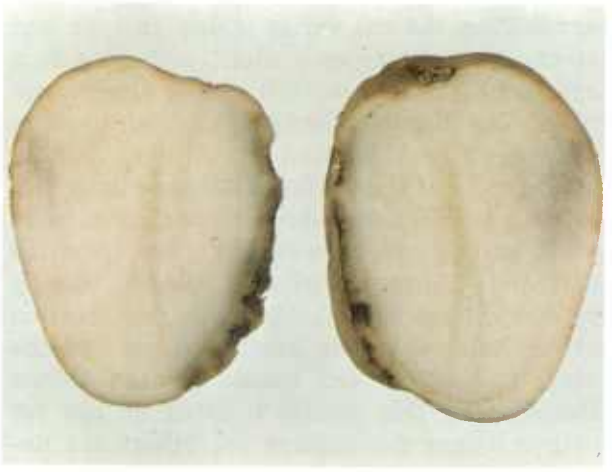

Figure 3.—Early blight lesions (cut-tuber view).

lower leaves followed by rapid wilting (fig. 4). Sometimes plants show the effect of infection slowly and succumb very gradually. A cortical decay of the underground stem may occur, and flecks appear in the stem pith. The woody tissues are yellow to brown, often from the base well into the top. Discoloration is more marked at the nodes.

Hot weather favors development of wilt disease but cool weather suppresses it. Wet soil and irrigation also favor disease development. If soil moisture is high, the foliage symptoms may never advance to the wilting stage, but leaves will show yellowing, rolling, and rosetting, sometimes accompanied by the development of aerial tubers. With medium soil moisture and heavy infection, the mottling becomes extended until the whole leaf surface is involved. Affected leaves turn yellow, dry, droop, and often hang from the stem by a thread. Sometimes an internal blackening of the small veinlets, the veins, and the petioles can be observed on surface tissue.

Vascular ring discoloration of roots and tubers may also develop. Stem-end tissues of infected tubers darken to a distinct brown color. Even if the fungus threads do not actually get into the tuber tissues, such discoloration may result when rapid killing of the stolon causes collapse of the stem-end tissues of the tuber.

Tuber rots.—Fusarium tuber rots are widespread; they cause heavy losses to potatoes in storage and markets. Infection usually takes place through wounds. The infection produces some form of dry rot, either as large sunken pockets or a wrinkled decay on the surface.

Fungi causing Fusarium tuber rots include: *Fusarium sambucinum* Fckl. f. 6 Wr., *F. caeruleum* (Lib.) Sacc., *F. trichotheciodes* Wr., and *F. avenaceum* (Fr.) Sacc.

Infected tubers usually develop a dry rot, but a moist rot may occur. The surface of the tuber is sunken or wrinkled (fig. 5). The rotted tissue is brown, gray, or black. Cavities often develop that contain yellow, pink, or red molds. In late storage, blue, black, purple, gray, white, or pink spore masses may develop on the surface of infected tubers.

Factors that contribute to Fusarium tuber rot include wounds, dirty tubers, high storage temperature and humidity, and tuber infection with other diseases, such as late blight, early blight, and common scab.

Seed-piece decay.—Fusarium seed-piece decay is caused by *F. sambucinum* f. 6 and *F. caeruleum.* When these organisms are present (fig. 6), poor stands of plants result, with subsequent reductions in yields. Seed-piece decay usually occurs when the soil is hot and dry and the cut surfaces of the seed pieces are not suberized properly.

Control

Wilt.—Fusarium wilt is difficult to control. To help reduce losses from this disease complex: (1) Plant disease-free seed potatoes; (2) treat the seed with a fungicide recommended

Figure 4.—Wilt caused by *Fusarium oxysporum* and *F. solani* var. *eumartii.*

Figure 5.—Fusarium tuber rot.

by local authorities; (3) practice crop rotation; and (4) follow good soil management, including use of proper irrigation practices.

Tuber rots.—To reduce losses from the potentially serious tuber-decay species, practice extreme care in harvesting and handling to prevent cuts and bruises. Store potatoes at a moderate temperature (15°C) and moderate humidity for a few days to hasten healing of wounds. Finally, store the potatoes at a cool temperature (5°) with moderate humidity and adequate ventilation.

Sebago and Keswick are highly susceptible to Fusarium tuber rots. Irish Cobbler and Kennebec are quite resistant.

Seed-piece decay.—To reduce losses from this disorder to a minimum (1) plant whole seed tubers, (2) handle cut seed properly to hasten suberization, and (3) treat seed pieces with a fungicide recommended by local authorities.

Jelly-End Rot

Causal agent: Fusarium solani f. *radicicola* (Wr.) Snyder & Hansen (*F. javanicum* Koord. var. *radicicola* Wr.); condition often sterile.

Jelly-end rot is a tuber defect caused by a *Fusarium* sp. and possibly by other organisms. Long-tubered varieties such as Burbank and Russet Burbank are particularly vulnerable. This rot occurs rather commonly on the Pacific Coast.

Jelly-end rot occurs only on tubers. Generally, the stem end of the tuber is affected. At harvesttime, the rot varies widely in type and extent; it ranges from a slight withering to a dry, wrinkled, sunken, and rather tough condition. The discoloration ranges from none to light brown or black and can involve half an inch or more of the tuber. Sometimes there is a soft and jellylike light-brown rot that extends as far as 1½ inches down the tuber from the stem end. However, affected tissues usually dry down, forming a sharp line of demarcation between the sound, or unaffected part of the tuber, and the rotted tissues. Stored tubers affected with this condition often do not rot further unless the storage conditions are unfavorable.

The exact environmental conditions that lead to the development of this rot are unknown. It is postulated that when soil moisture is deficient late in the growing season, the plant may actually withdraw water from the stem end of the tuber. This brings about a sunken, withered condition favorable for the entrance of weakly parasitic organisms. Rot soon follows. No single organism has been consistently associated with jelly-end rot, which develops in the crop

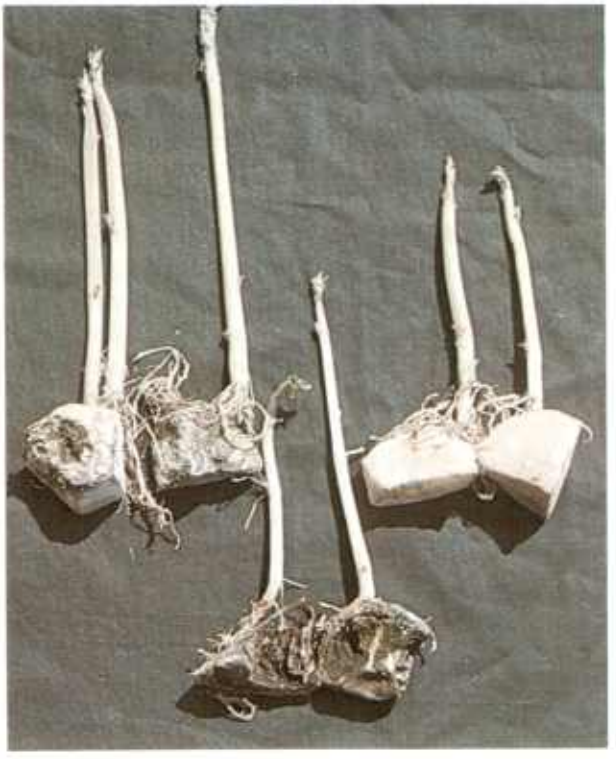

Figure 6.—Fusarium seed-piece decay caused by *F. sambucinum* and *F. caeruleum*.

without any apparent relation to the condition of the seed potatoes at time of planting.

Control

No control is known for jelly-end rot. The disease can be avoided to a certain extent by maintaining uniform and adequate supply of moisture in the soil throughout the growing season.

Late Blight

Causal agent: Phytophthora infestans (Mont.) D By.

Late blight is an old and widely known fungus disease of potato. It is a historic disease, responsible for the potato crop failure that resulted in the Irish "famine" in the 1840's.

This disease affects leaves, stems, and tubers. It appears on the leaves as pale-green, irregular spots (fig. 7*A*). In moist weather those spots increase rapidly in size, their centers die and turn dark brown or black. Often the spots have a purplish tint. On the lower sides of the leaves a white mildew ring forms around the dead areas. In dry weather the water-soaked areas turn brown, dry up, and die. Necrosis often begins at the tip of the leaf and in severe cases, entire leaves are killed.

Stems and petioles turn brown when infected. Under humid conditions, the entire vine may be killed and blackened in a short time. In warm, moist weather late blight spreads rapidly, and all plants in a particular field may be killed in a few days. The diseased and decaying plants give off a fetid odor that becomes very pronounced in severely attacked fields.

Tubers are readily infected while in the soil by rainborne spores from blighted tops and at harvest by contact with blighted foliage. Initial tuber infection results in a shallow, reddish-brown dry rot (fig. 7*B*) that spreads irregularly from the surface through the flesh like the diffusion of a brown stain. At first the affected tissue is dry and firm with a somewhat caramelized, sugary texture. Soft rots often follow the late blight rot and completely destroy the tuber. Some blighted tubers rot in the field, especially those in low, wet spots. Also, potato tubers frequently become inoculated with spores from infected plants during the harvesting operations and are later affected by late blight tuber rot while in storage. In storage the disease is typically a dry rot, forming irregularly shaped, sunken patches. Under conditions of high humidity and temperatures, this dry rot

Figure 7.—*A*, Potato leaf infected with late blight; *B*, tuber showing symptoms of late-blight tuber rot.

may involve the entire tuber. At low storage temperatures, the patches usually remain rather firm; frequently they have a metallic tinge, especially at the border of healthy tissues.

Phytophthora infestans thrives under cool, moist conditions. This fungus produces conidia that germinate at 24° C by producing germ tubes that penetrate the leaf through the stomata. The fungus also germinates at lower temperatures by producing numerous zoospores, often called swarm spores. Zoospores are produced abundantly at 12° in the presence of moisture. These zoospores then germinate readily when the temperature rises to 15° to 22°. The most favorable conditions for a severe outbreak (epiphytotic) of late blight disease are night temperatures of about 13° with rain or heavy dew, followed by day temperatures of 15° to 24° with high humidity in the form of rain, fog, or lingering dew.

The perfect, or oospore, stage of *P. infestans* has been found in Mexico. If this stage occurs in the United States, its role in the disease cycle appears to be insignificant. However, this fungus is extremely mutable and more than 15 pathogenic races have been identified. These races are designated by number, singly or in combination, depending on the R genotypes they attack. Six dominant *demissum* genes for resistance have been noted.

Sources of disease-causing inoculum are several. This fungus overwinters in infected tubers. Cull piles on farms and near potato storage houses are a primary source of infection because these cull piles often contain a high percentage of late blight-infected tubers; the young shoots from such tubers are also infected with the fungus. Such infections generally develop during the early part of the growing season before most growers have begun to spray. The spores may be carried by the prevailing winds to the neighboring fields and there infect the emerging potato plants. Early infections spread rapidly when drifting mist, fog, and wind-blown rains are prevalent. Late blight, when first observed in the field, often appears as small, isolated spots on the top leaves or stems of the potato plants. The disease may appear in this form at about the same time in a number of fields distributed over a large area. In these instances, the disease is dispersed by the wind.

The late blight fungus may also grow from a diseased seed tuber up the stem to the surface of the soil, sporulate, and cause infection of the foliage. However, under normal field conditions the diseased shoots often die before they emerge from the soil, especially if the seed piece has been planted deeply.

The number of seed tubers that develop infected shoots is often small. However, infected shoots may still be a serious source of infection because they may emerge through the soil, die 1 or 2 days later, and leave only a small black tuft of decaying tissue. Because such tissue is difficult to locate, the vacant spot is apt to be attributed to a missing hill. However, in the brief period that the infected sprout is above ground, the spores produced on it have an opportunity to spread to one or more adjoining plants and thus provide an infection spot in the field. Under favorable conditions, the fungus can then spread readily throughout the field.

Control

Extensive research has led to development of effective control methods for this disease. A combination of these control measures is followed by commercial growers. Recommended practices for control of late blight are (1) plant only healthy seed potatoes, (2) destroy or eliminate all potato cull piles, (3) spray or dust with an effective fungicide, (4) kill infected potato tops before harvest or spray them with a vine killer to prevent tuber infection during harvest, (5) control powdery scab to reduce susceptibility to late blight infection, and (6) plant resistant varieties.

For many years, bordeaux mixture was the standard fungicide used to control late blight. However, although bordeaux mixture is effective, it is also phytotoxic. "Neutral," or fixed copper, fungicides have partly replaced bordeaux mixture because they are less phytotoxic, more convenient to prepare, can be used as a dust formulation, and are fairly effective in controlling late blight. In recent years the neutral fungicides have, in turn, been largely replaced by the organic fungicides that are effective against both early blight and late

blight. Maneb provides excellent control of both diseases and is widely used by potato growers.

Some States provide a late blight forecasting service as an aid to growers in timing their spray or dust applications. Forecasts are based on records of temperature, rainfall, relative humidity, or a combination of these factors, and the probability that late blight will develop under the prevailing environmental conditions if inoculum is present.

Although dusting is not quite as effective as spraying under severe blight conditions, it has some advantages. Dusting affords rapid plant coverage and less soil compaction. Dusting also requires less expensive equipment and no water supply. Aerial spraying is gaining in popularity in some areas. This type of spraying should reduce the danger of transmission of certain sap-transmissible viruses.

Resistant varieties can also play a major role in the control of this potential disastrous disease. Most resistant varieties are not immune to late blight but possess varying degrees of resistance to the various races of the pathogen. Among the older varieties which possess some resistance to the common race, Race O, of the fungus are Sebago, Russet Sebago, and Sequoia. More recent introductions usually include a higher degree of resistance to the common race of the fungus, and many of them possess some resistance to other races also. Late blight resistant varieties released in the United States are Alamo, Boone, Catoosa, Cherokee, Chieftain, Delus, Kennebec, Merrimack, Onaway, Pennchip, Plymouth, Pungo, Reliance, Saco, Tawa, and Wauseon. Blight-resistant Canadian varieties include Keswick and Fundy. A number of late blight-resistant varieties have also been developed in Mexico. They are Anita, Bertita, Conchita, Dorita, Elenita, Erendira, Florita, and Greta.

Leak

Causal agent: Pythium spp., mainly *P. debaryannum* Hesse, *P. ultimum* Trow.

Leak is the name given to a water rot or a watery wound rot of potato. The condition affects the tuber and is caused by species of a soil fungus, *Pythium,* and possibly by several species of *Phytophthora* that are closely related to the species that causes late blight. The disease is a problem only when potatoes are harvested and moved during warm weather. This situation would apply to the early crop of Idaho and Washington and to the crop grown in the upper San Joaquin Valley of California. Leak has also been found in Virginia, on Long Island, N.Y., in Rhode Island, and in New Hampshire.

Pythium spp. are soilborne organisms that enter tubers through wounds. The most characteristic symptom of leak is the extremely watery condition of affected tissues. The water is usually held by the disintegrated tissues. When pressure is applied, a yellowish to brown liquid is readily expelled.

When a tuber affected by leak is examined, external or internal fungus growth is rarely discernible. The cavities caused by Pythium infection are not lined by white or brightly colored molds as are the cavities in the soft types of Fusarium rot, a disease that may resemble leak infection. The colors of leak-affected tissues more closely resemble tissues affected by blackheart, but blackheart-affected tissues do not become soft, watery, and granular as do tissues infected with *Pythium* spp.

Another major characteristic symptom is the granular condition of the diseased tissues. Externally, an affected tuber may show discoloration ranging from a metallic gray in red-skinned varieties to brown in white- and dark-skinned varieties. Internally, the affected tissues are creamy at first. When the tuber is cut further, and in the later stages of the disease, the tissues soon turn tan or slightly reddish, then brown, and finally inky black. The diseased areas are sharply set off from the healthy tissue and often only a shell of sound tissue remains.

Infection of tubers occurs during hot weather and frequently occurs in tubers that are damaged by sunburn and sunscald, especially in tubers allowed to remain in or on hot soils after being dug. At temperatures between 16° and 32° C, lesions become visible within 36 hours after infection. At temperatures above 10°, leak lesions are usually invaded by bacteria. These bacteria check the growth of the leak

organism and produce foul-smelling, sticky, or slimy decays.

Control

To control leak, harvest tubers when they are mature. Harvest during cool weather and keep the tubers as cool and dry as possible during early stages of transit. Store harvested tubers at a cool temperature. Use care in handling to avoid bruising and injury to the skin.

Phoma Tuber Rot

Causal agent: Phoma exigua Desm. var. *foveata* (Foister) Boerema
=*P. foveata* Foister
=*P. solanicola* Prill. & Del. (*P. tuberosa* Melhus, Rosenbaum & Schultz)
=*P. solanicola* f. *foveata* (Foister) Malcolmson

Phoma tuber rot is a disease of potato tubers in storage. In the United States this disease is called button-hole rot, but in other countries, it is usually called gangrene.

Tuber infection occurs primarily through wounds, but infection can take place through the eyes and the lenticels. Seed pieces that do not heal properly may also be attacked. The surface of infected tubers becomes brown to gray and this discoloration darkens with age. The skin becomes papery and often tears, which gives it the appearance of a ragged buttonhole. Black dots (pycnidia) may develop on the tuber in late storage. The affected tissue is dark gray, dry, and powdery.

In Europe *P. exigua* var. *foveata* attacks stems. The infection causes brown lesions that enlarge and cause the older decayed tissue to turn white. This effect is accompanied by a stalk break. Yield is also reduced.

Cool, dry storage conditions that are unfavorable for healing of wounds favor tuber infection. The infection may also start in lesions caused by other fungi, such as powdery scab.

Control

Handle the potatoes carefully to prevent wounding and store them at 18° C or higher for 10 days to promote rapid healing of wounds. Cariboo is a new resistant variety.

Pink Rot

Causal agent: Phytophthora erythroseptica Pethyb.

The pink rot disease of potato has been reported in the United States, Canada, and Australia, and several European countries. The causal organism is a soilborne fungus that causes a late-season wilt of plants and a tuber rot when the weather is cool and soil conditions are wet. When tubers are affected, this disease is commonly referred to as pink rot or water rot.

When plants are attacked by this fungus, the symptoms include a late-season wilt and a decay of the lower part of the stems. The fungus, however, primarily attacks the tuber. Infection usually starts at the stem end but may take place through lenticels and wounds. Surfaces of affected tubers have a dull, dark-brown, water-soaked appearance. Affected tissue becomes wet and rubbery, and has a dirty-white color that later turns pink when the cut tuber is exposed to air.

Control

Where pink rot is a problem, supply irrigation and good drainage to avoid accumulation of excessive moisture. During digging, remove infected tubers before the crop is stored or shipped. Avoid wounding tubers and store them under relatively cool, dry conditions.

Powdery Mildew

Causal agent: Erysiphe cichoracearum DC. ex Mérat

Powdery mildew is a disease on potato and other crops in temperate regions of the world. Powdery mildew of potato is caused by an obligate parasite, *Erysiphe cichoracearum.* This fungus is characterized by a spore form with simple or irregularly branched appendages and a cleistothecium that contains several asci. This sexual stage is the only stage of the fungus that occurs in Washington State; most commonly only the conidial stage is present, which makes accurate identification very difficult. The fungus has been reported on potatoes growing in greenhouses in New Jersey and Kentucky and in England. In the field, it has been re-

ported in Washington State and in England, Argentina, and Palestine. The disease usually occurs late in the growing season and causes little or no harm. However, substantial losses have been reported in Palestine.

Powdery mildew on potatoes is confined almost entirely to the petioles and stems of terminal growth. The conidial growth is a white to grayish growth of mycelium, conidiophores, and conidia. The conidia are usually borne in chains and are readily detached from the conidiophore and from each other. They are dispersed by air currents. The growth of *Erysiphe* on potato leaves is somewhat compact and lacks the glistening moisture usually found with the sporulation of *Phytophthora.*

In England, small patches of *E. cichoracearum* develop on the leaf surfaces but not on the petioles or stems. It is possible that different species are involved in the powdery mildew infestations in Palestine.

Control

Sulfur sprays can be applied, but the benefits derived may be insufficient to justify their use.

Rhizoctonia Canker (Black Scurf)

Causal agent: Rhizoctonia solani Kühn

Rhizoctonia canker, or black scurf, is a very common and serious disease of the potato. The disease is present in every potato-growing area. *Rhizoctonia* has a wide geographic distribution and a wide host range. Other common names for this disease are sprout canker, stem canker, rhizoc, and rhizoctonose. An expression often applied to the disease is "the dirt that won't wash off." The proper scientific terminology for the fungus is in debate. The imperfect (mycelial and sclerotial) stage of the fungus is usually called *Rhizoctonia solani*; the perfect (basidial) stage is commonly cited as *Pellicularia filamentosa* (Pat.) Rogers and since 1956 as *Thanatephorus cucumeris* (Frank) Donk. This perfect stage occurs on green stems near the surface of the ground when conditions are humid. The affected stems become covered with a white, dirty-gray or purplish feltlike mycelial mat (fig. 8*B*) on which tiny basidia and basidiospores are produced.

The fungus can overwinter as sclerotia on infected tubers or as mycelium or sclerotia in infested soils. Because of its wide host range and

Figure 8.—Rhizoctonia infection: *A*, Rolling of upper leaves of potato plant caused by Rhizoctonia infection of roots and underground portion of stems; *B*, lower portion of potato plant infected with *Rhizoctonia*. Note the mycelial mat and the aerial tubers.

because of its longevity in the soil, this fungus can persist for several years or more in most infested soils. The conditions favoring Rhizoctonia infection are high moisture, cool soil temperatures, high soil fertility, and a neutral to acid soil with a pH of 7 or less.

Potato plants attacked by *Rhizoctonia* are characterized by a general lack of vigor when the attack is mild. An uneven stand of weakened plants results when young shoots and stems are girdled. Few top symptoms appear if the underground part of a stem is only partly girdled by the fungus. When girdling is complete, the top symptoms resemble those of psyllid yellows—the foliage curls and turns pinkish to purplish. Often small, green or reddish aerial tubers are formed as a result of the interference of starch translocation (fig. 8*A*, 8*B*). Either complete or partial girdling reduces the number of normal-size tubers or limits the yield to a few small tubers set close to the surface of the soil. If exposed to light, the tubers of many varieties will turn green.

Long, brown lesions can occur on affected shoots, stems, and stolons (fig. 9*B*). Sprouts often decay before they reach the surface of the soil, or the roots may be severely killed back. Nonproductive adventitious rootlets and secondary stolons then develop. Rosettes or clusters of sprouts are formed, none of which emerges from the soil (fig. 9*C*).

The most common symptom of Rhizoctonia canker is the presence of numerous, hard, small, dark-brown or black resting bodies that are called "sclerotia" on the surface of mature tubers (fig. 9*A*). These sclerotia vary in size from a pin dot to half a pea. Sometimes these masses, actually threads of the fungus, can be mistaken for soil until it is found that they do not wash off. They do no harm to the tubers except to detract from their appearance. However, if infected tubers are used for seed, the mycelial threads from the sclerotia will infect shoots, roots, and other parts of the growing plant. Other tuber symptoms include russeting, cracking, knobbiness, and dwarfing. This fungus has also been implicated as the causal agent of "lenticel spot," a condition in which dry, blackened pits or lesions occur at the lenticels or "eyes" of affected tubers.

Control

Rhizoctonia canker is very difficult to control. To help check this disease, use disease-free seed or disinfected seed and practice crop rotation with cereals and grasses. Shallow covering to hasten emergence is also recommended.

Soil treatment with PCNB has shown promise for control. This product can be applied in broadcast application, mixed with the top 4 inches of soil, or used in a modified broadcast row-application procedure.

Scab, Common

Causal agent: Streptomyces scabies (Thaxt.) Waks. & Henrici

Common scab is a disease that attacks tubers. It is known to exist in every potato-growing area of the United States.

On tubers, one type of scab produces brownish spots that are small at first but later enlarge. The resulting lesions are large and very corky (fig. 10). Frequently they extend below the tuber surface and leave deep pits when the corky tissue is removed. This type lesion is known as the pitted type of scab. Deep or pitted scab lesions are roughly circular, 1 to 5 mm in diameter, and are surrounded by corky tissue. Thin-skinned varieties are susceptible.

In another form of scab, known as surface scab, the lesions appear as small russeted areas so numerous that they almost cover the entire tuber surface. With this type of lesion, slight protuberances with depressed centers may form. These lesions are covered with a small amount of corky tissue. Russet varieties are susceptible to this form of scab.

Common scab is particularly severe in alkaline soils above pH 7 or in soils that are about neutral (pH 7). The optimum soil pH for *S. scabies* is 6 to 7.5. However, some races are capable of growing and causing infection at pH 5 or below. Infection that takes place under this unusual condition is sometimes referred to as uncommon scab or acid scab to distinguish it from common scab.

Common scab lesions are sometimes confused with abnormally enlarged lenticels. Also, some flea beetle injury, caused by the larvae of three species of the flea beetle (*Epitrix cucumeris* (Harris), *E. subcrinita* (LeConte), and *E. tu-*

beris Genter), may resemble the external appearance of the first stages of common scab infection. However, when the pustules or furrows made by the flea beetle larvae are cut through, tough splinters of corky tissue are found extending perpendicularly one-fourth to one-half inch into the tuber. Often these furrows caused by the flea beetle are infected sec-

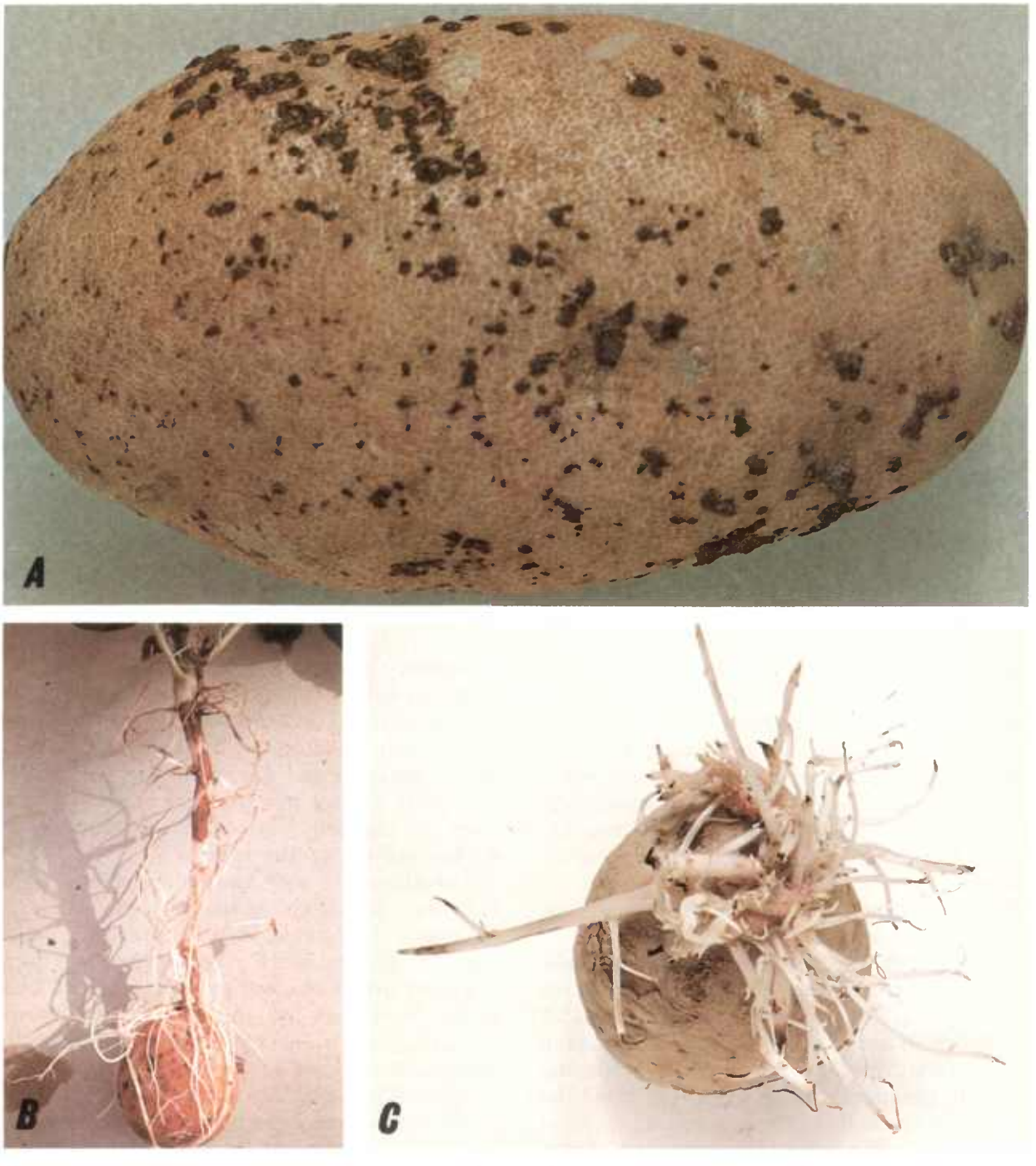

Figure 9.—Rhizoctonia infection: *A*, Sclerotia on surface of the tuber; *B*, underground stem of young potato plant girdled with the fungus; *C*, cluster of sprouts infected with *Rhizoctonia*.

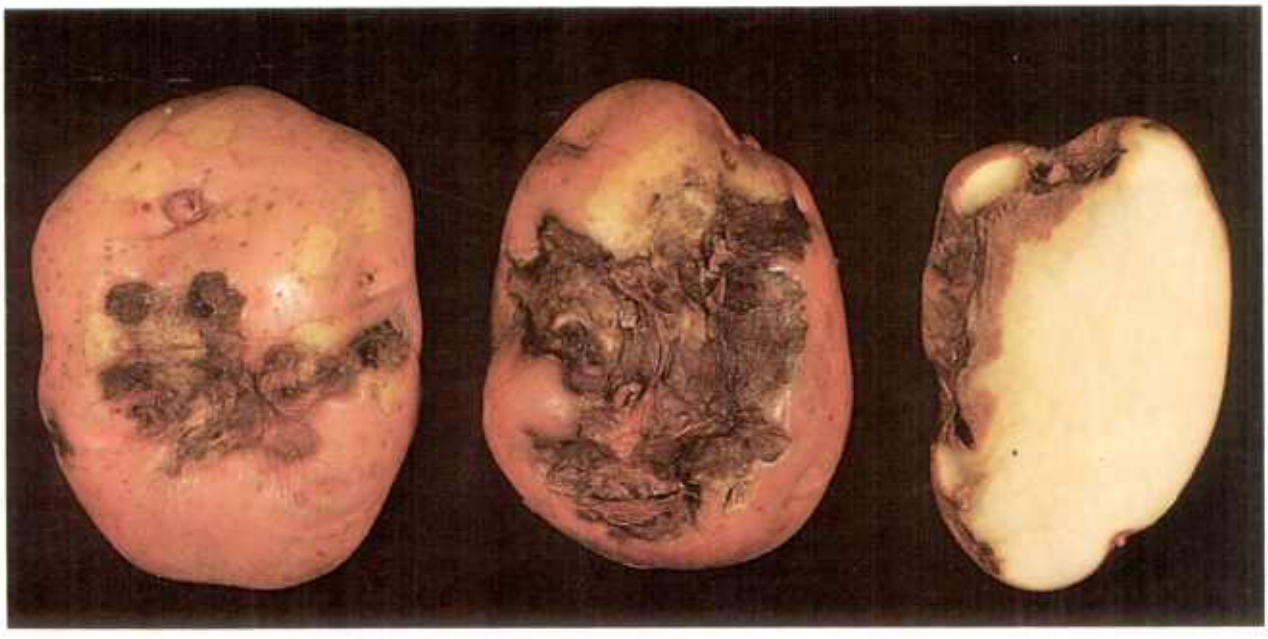

Figure 10.—Potato tuber showing severe infection with common scab.

ondarily with the Rhizoctonia canker and the common scab organisms. These infections deepen the furrows and the insect-feeding scars.

Control

The most important method of scab control is to plant resistant varieties. The newer, scab-resistant varieties include Alamo, Arenac, Antigo, Avon, Blanca, Catoosa, Cayuga, Cherokee, Chieftain, Chinook, Hi-Plains, Menominee, Navajo, Norgold Russet, Ona, Onaway, Ontario, Pennchip, Platte, Plymouth, Pungo, Redskin, Reliance, Sable, Seneca, Shoshoni, Superior, Tawa, Wauseon, and Yampa. Russet-skinned varieties, such as Early Gem, Russet Burbank, Russet Rural, and Russet Sebago, are partly resistant.

Soil treatment with formaldehyde may be effective under some conditions.

Other control measures are (1) plant scab-free or disinfected seed potatoes in scab-free soil, (2) adjust or maintain soil pH at 5 to 5.2 in soils which are naturally acid, (3) choose the proper fertilizers, preferably acid-producing ones, and the proper cover crops, (4) avoid the use of too much lime, fresh manure, or wood ashes (if lime is needed, dolomitic lime should be applied following potatoes in the rotation), and (5) reduce the inoculum potential in the soil with long rotations. Where a history of scab exists, exclude other hosts, such as table beets and sugar beets, from the rotations.

Scab, Powdery

Causal agent: Spongospora subterranea (Wallr.) Lagerh.

Powdery scab is a fungus disease that attacks the tuber. It is primarily a disease of the Northern States and Canada, but it also occurs occasionally in some of the Southern States where potatoes are grown during the winter and early spring months. The causal fungus lives in the soil, and infection takes place during growth of the tubers.

The disease in part resembles common scab. However, the individual lesions are more nearly circular and, as a rule, they are smaller in diameter. In the immature stage of powdery scab the spots are closed, somewhat raised and blisterlike, and dark on the outside and brown or olive brown on the inside. When fully mature, the lesions are roundish, raised, open pustules. These pustules are filled with a brown, powdery mass of spores and broken-down tissue and are surrounded by fringed remnants of the skin (fig. 11). Small galls sometimes occur on the roots of affected plants. Infected tubers may

Figure 11.—Potato tuber infected with powdery scab.

develop dry rot in storage. This disease also predisposes tubers to late blight tuber rot. *Spongospora subterranea* has been reported to transmit a virus disease called mop top.

Control

Powdery scab is not satisfactorily controlled by seed treatment. Because the disease is a problem in cool, wet soils, avoid these growing conditions. Avoid infested soil, and also, use disease-free seed potatoes and practice long rotations.

Sclerotinia White Mold (Stalk Break)

Causal agent: Sclerotinia sclerotiorum (Lib.) D By. (=*Whetzelinia sclerotiorum* (Lib.) Korf & Dumont).

Sclerotinia white mold is one of the most widespread of the diseases that attack vegetables. It is the cause of a watery soft rot. Fortunately, potato is seldom attacked by *Sclerotinia*. However, it has occurred in the South where it has been of major concern to potato growers, particularly when conditions favor its development. These conditions include cool, rainy weather, prevalence of fogs, heavy dews, and shade.

The fungus attacks the stem of potato plants and the stems of soft, succulent plants. It causes a light-colored soft rot. The disease first appears on a stem as a water-soaked lesion, then the white mycelium of the fungus grows over the stem surface. All parts of the stem except the outer woody tissues are destroyed, and the interior becomes packed with the mycelium and black sclerotia. The plant wilts and finally dies.

The fungus is dormant during the winter and during warm, dry weather at other seasons. Sclerotia survive in old plant debris or in the soil.

Control

To control Sclerotinia white mold, practice good sanitation and crop rotation. The sclerotia can be killed if the land is flooded with water and left inundated for about 5 weeks.

Sclerotium Rot (Southern Blight)

Causal agent: Sclerotium rolfsii Sacc.

Sclerotium rot, or southern blight, is a disease of potatoes and various truck crops in the southern part of the United States. The disease-causing organism is a soil-inhabiting fungus that, under favorable moisture and temperature conditions, attacks a wide range of hosts.

This fungus attacks potato vines in the field, most frequently at the surface of the ground. During the heat of the day the young leaves wilt, but they recover at night. As the wilting progresses, the foliage turns yellow and the

leaves fail to recover. Eventually the leaves die, the stems lose color, and the entire plant dries out and dies.

At the base of affected stems, sunken, discolored areas are produced, usually just below the surface of the ground. These areas become water soaked and finally become covered with a white mycelial mat of the fungus. This threadlike fungus mat on the underground stem surface is a major characteristic of the disease. The mat is a very fine, silky, growth, developing in a radial fashion and forming fanlike structures. These white fans may be found on the soil as well as on plant parts; they are sometimes found on crates and bags. Sclerotia, small fruiting bodies that are white at first but later become dark, are produced in the mats. These sclerotia are about the size, shape, and color of radish or mustard seeds.

Sclerotium rot also attacks the tuber, causing a decay and rot. In the early stages of development, the rot is white and almost odorless; in later stages it becomes yellow. When the attack is mild, slightly sunken spots only are produced. However, the decay often develops rapidly and involves the entire tuber, producing a slimy condition.

Control

Because the fungus causing Sclerotium rot is soilborne and attacks a wide range of vegetable crops, control is difficult. The disease is usually of minor importance, and the fungus spreads very slowly in the soil.

Because serious losses of potatoes frequently occur in transit, precautionary measures are to (1) avoid harvesting and packing operations during wet weather, and (2) dry the tubers thoroughly before crating or bagging.

Silver Scurf

Causal agent: Spondylocladium atrovirens Harz.

Silver scurf is a common disease that attacks potato. It is usually a disease of minor importance, but, when potatoes are grown on muck soil, the blemishes produced by this parasite may cause reduction in grade or preclude seed certification.

The silver-scurf pathogen appears to be strictly a tuber disease. The tubers become susceptible at time of maturity while they are still in the soil and remain susceptible throughout the storage period. Light- to dark-brown spots develop on the tuber surface. In severe cases, the spots coalesce and cover large areas of the tuber (fig. 12). Infected areas appear silvery and glassy when wet, attributes that have given this disease the name "silver scurf." Sloughing of the outer skin occurs because the suberized cells are loosened. These affected areas shrivel and shrink. Under severe conditions, this destruction of the skin will interfere with market and seed quality. The color in red-skinned varieties may be completely destroyed by the disease. High humidity favors disease development.

Control

Plant disease-free seed tubers and practice crop rotation to reduce the incidence and severity of silver scurf. Harvest tubers as soon as they are mature and store them in a cool, dry place.

Skin Spot

Causal agent: Oospora pustulans Owen & Wakef.

Skin spot is the name given to a normally inconspicuous disease of the tuber that can become important if it is severe enough in the

Figure 12.—Potato tuber infected with the silver scurf organism.

region of the eyes to prevent sprouting of the seed piece.

Small, dark pustules, about 1 mm in depth, develop on the tuber (fig. 13). They are dark brown on the outside and olive brown inside. These pustules have been confused with those caused by the early stages of powdery scab. A brown cortical rot of the underground stem also occurs. The fungus overwinters in seed tubers and in infested soil.

Control

To minimize severity of this disease, plant disease-free seed tubers and practice crop rotation.

Verticillium Wilt

Causal agent: Verticillium albo-atrum Reinke & Berth.

Verticillium wilt of potato is a disease that can cause serious losses in major potato-production areas. In the Pacific Northwest, it is often referred to as early dying. When warm weather occurs early in the growing season, Verticillium wilt also appears earlier.

Verticillium albo-atrum is a soilborne organism that has a wide host range, including solanaceous crops, such as tomato, eggplant, and potato, and nonsolanaceous plants, such as small fruits and shade trees. The fungus can overwinter in soil adhering to the surface of potato tubers, either as mycelium or as microsclerotia, and it may also overwinter as mycelium inside the tuber. In culture, this fungus produces a dark mycelial type of growth, designated "D," or it produces a microsclerotial type of growth, designated "M." Type M is sometimes classified as *V. dahliae* Kleb., a separate species. In distribution type D is believed to be indigenous to Maine and the type M is believed to be indigenous to Idaho. Type D is more prevalent in Maine and type M is more prevalent in Connecticut, but both types are found in Maine. Both types are distributed about equally in Ontario, Canada, but type D is more pathogenic.

The plant symptoms of Verticillium wilt are wilting, yellowing, and premature death (fig. 14). Attacked plants may wilt rather suddenly and die in a comparatively short time, or the plants may succumb gradually. Some curling and rolling of the leaflets and a tipburn may occur. Vines so affected die prematurely, but the stem remains upright except for the tips that may droop. Yellowing and dying of the leaves proceed from the base of the plant upward until often only a cluster of green leaves remains at the top. On very hot days, yellowing

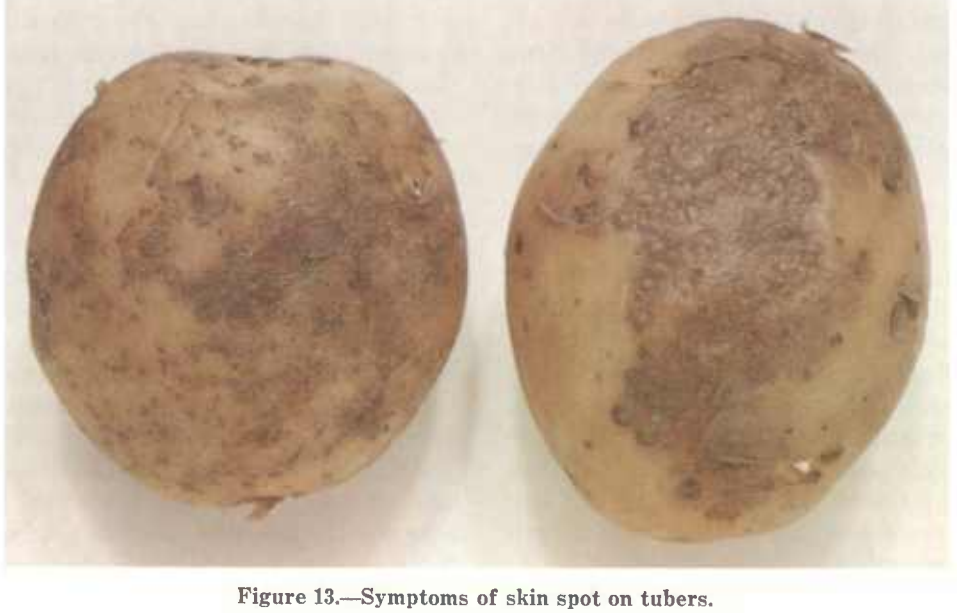

Figure 13.—Symptoms of skin spot on tubers.

Figure 14.—Potato field showing severe Verticillium wilt symptoms.

is preceded by wilting of the leaves and even of the stem.

The stem interiors of infected plants are always discolored. The woody portion of the stem (xylem) becomes yellow then turns a reddish brown that often can be seen from the base to well into the plant top. Sometimes when the top leaves wilt and die before the lower ones, the plant looks as if it were scalded. To confirm the diagnosis of wilt, the xylem-ring discoloration can be observed by cutting away the bark at the base of the stalk or by cutting the stem at a sharp angle about ground level (fig. 15*A*). If the plant suffers from wilt, the woody tissue beneath the bark is brown instead of the white or faint yellow color of healthy tissue. The discoloration may extend all the way around the stem or may be confined to one side. All the fine feeding roots and the bark of the larger taproot become entirely decayed.

Tubers from affected plants may show a brown or black discoloration of the vessels at the stem end (fig. 15*B*). However, do not use stem-end discoloration alone to diagnose wilt. Several other factors, such as freezing injury, vine killers, and stem-end browning, can produce a xylem-ring discoloration. When wilt is severe, cavities may develop inside the tubers.

As a complication to wilt-induced tuber problems, a pink or tan discoloration, called pink eye or brown eye, sometimes develops around the tuber eyes or as blotches on the surface of affected tubers (fig. 16). This discoloration, especially common in the Kennebec variety, may be confused with mild late blight infection. Under cool, dry storage conditions these pink areas turn brown and dry up.

An association between wilt and pink eye has been suggested, but the exact relationship between the two diseases has not been clarified. A bacterium, *Pseudomonas fluorescens* (Trevisan) Migula, has been isolated from the discol-

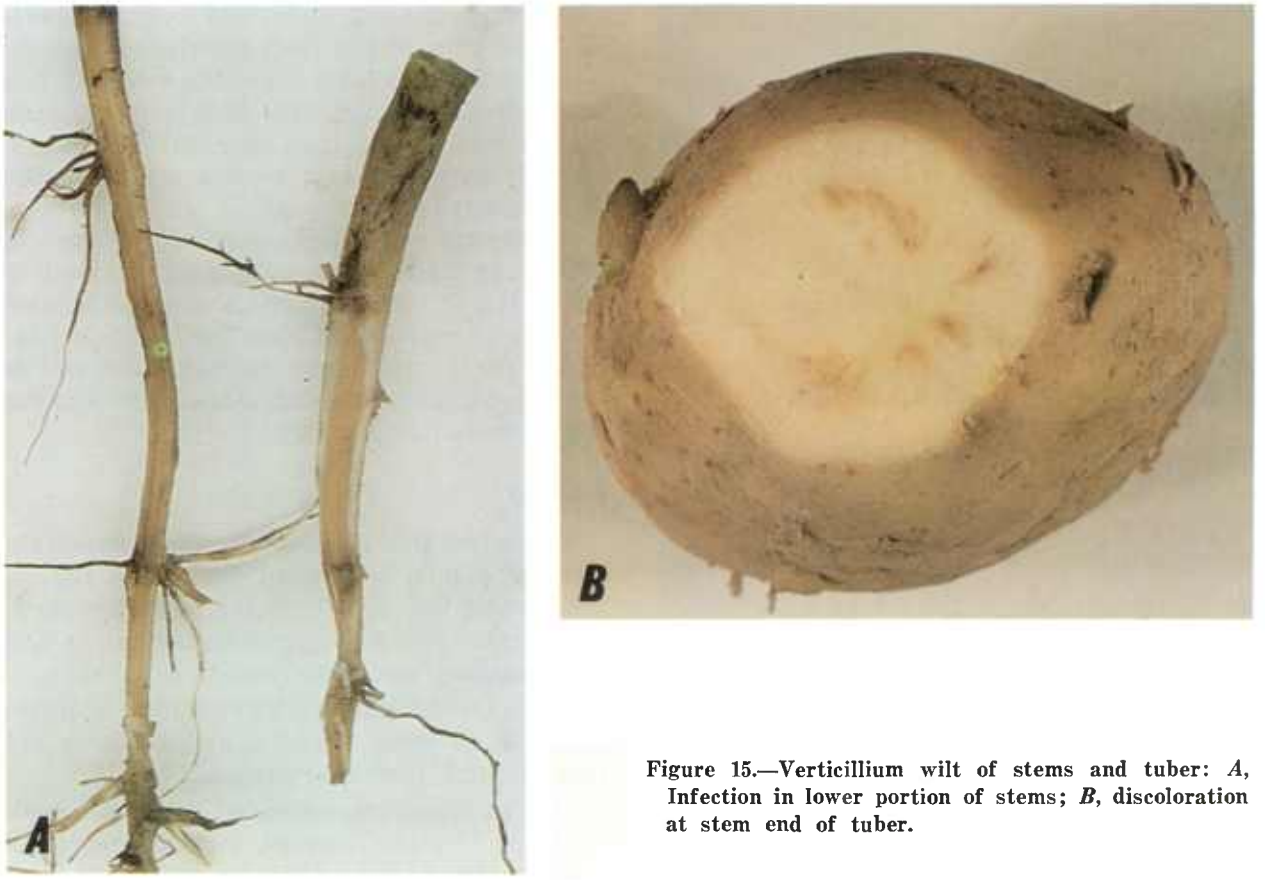

Figure 15.—Verticillium wilt of stems and tuber: *A*, Infection in lower portion of stems; *B*, discoloration at stem end of tuber.

ored areas that occur in pink eye, and the pink-eye condition, as associated with the presence of this bacterium, can occur even in the absence of Verticillium infection. When *P. fluorescens* is present, infected tubers break down in storage at temperatures above 7° C, and the bacterial infection spreads from diseased to healthy tubers in storage.

The severity of Verticillium wilt may increase where the lesion nematode (*Pratylenchus penetrans* (Cobb) Chitwood & Oteifa) is prevalent.

Control

To control Verticillium wilt (1) avoid planting infected seed and planting in infested soil, (2) practice crop rotation, excluding susceptible crops from the land, (3) control weeds that may serve as hosts for the fungus, and (4) plant resistant varieties.

Vapam, used as a soil treatment, has reduced the amount of damage caused by wilt.

The older American varieties resistant to Verticillium wilt are Houma, Menominee, Ontario, Russet Rural, Sequoia, and Yampa. New resistant varieties include Cariboo, Hunter, Ona, Red Beauty, and Shoshoni. Reliance is tolerant of the disease.

Wart

Causal agent: Synchytrium endobioticum (Schilb.) Perc.

Potato wart, variously known as black wart and canker, was first recognized in Europe and was introduced into home gardens in an eastern nonseeding-growing area of the United States about 1912. It did not become widespread in the United States but was limited to a few areas of southern Pennsylvania, western Maryland, and northern West Virginia. Extensive soil treatment of infested areas and monitoring of subsequent home

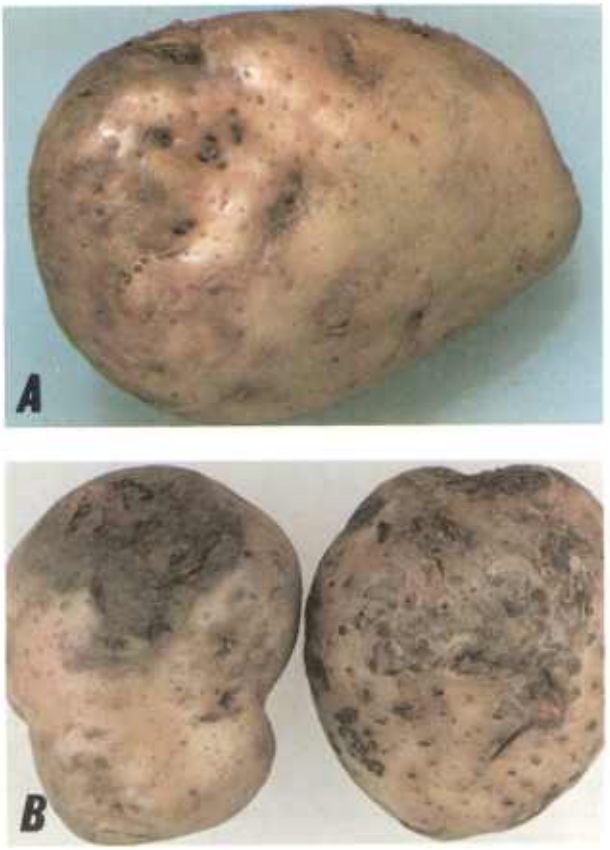

Figure 16.—Mild (A) and severe (B) symptoms of "pink eye."

garden potato crops indicate the wart organism has been eradicated from infested areas. Wart is, however, a most destructive disease of the potato in infested soils in Newfoundland, Canada.

This disease is characterized by warty outgrowths on all parts of the potato except the roots. The warts occur on buds, the bases of stems, the stolons, and on the tubers. On occasion, they occur even on leaves and flowers. These warts are soft, puffy, and roughly spherical. They vary in size from the size of a pea to large masses that cover the entire tubers. When warts occur underground, they are white or pinkish when young, then turn darker with age. The aboveground warts become green because of chlorophyll development.

Synchytrium endobioticum can live in the soil for a long time. When wart-infected tissue is left in the soil, it soon breaks up and liberates millions of spores that leave the land badly infested for years. When wart-infested soil is kept in sod, the fungus can remain active in the soil for more than 20 years.

Control

To control this disease effectively, eradicate diseased plants, heed plant quarantine regulations, avoid the use of infested fields, use certified disease-free seed, practice sanitation and crop rotation, and plant resistant varieties.

In the United States, Irish Cobbler, Katahdin, Mohawk, Sequoia, Mesaba, and Norkota are immune, and Houma is resistant.

The varieties recommended for Newfoundland are Urgenta, Sebago, and Kennebec. In Newfoundland, Katahdin is resistant, but Houma, Mohawk, and Sequoia are very susceptible.

Varieties that are resistant to the potato wart fungus in Europe are not resistant in Newfoundland. The ability of the fungus to parasitize other solanaceous species, such as nightshade (*Solanum nigrum* L.), the true bittersweet (*S. dulcamara* L.), and tomato (*Lycopersicon esculentum* Mill.) and the variation in resistance in different geographic locations, indicate that probably different physiologic races of the pathogen occur.

BACTERIAL DISEASES

Single-celled organisms that do not form reproductive spores and that reproduce by fission (splitting) are termed "bacteria." These organisms are microscopic. They usually thrive under warm, moist conditions and are capable of multiplying rapidly. Bacteria cause wilting or soft rot of potato plants and tubers. They normally gain entrance to the plant via wounds or natural openings.

Four major bacterial pathogens attack potato, which serves as their host plant. These pathogens are found in three families of the Order Eubacteriales: Pseudomonadaceae, Enterobacteriaceae, and Corynebacteriaceae. The

representative potato genus and species for the Pseudomonadaceae is *Pseudomonas solanacearum;* for the Enterobacteriaceae, *Erwinia carotovora* and *E. phytophthora.* These bacteria are rods that are motile and gram negative. *Corynebacterium sepedonicum,* representing the family Corynebacteriaceae, is nonmotile and gram positive.

The potato diseases caused by each of these pathogens and suggested controls are listed in table 1.

Bacterial Soft Rot

Causal agent: Erwinia carotovora (L. R. Jones) Holland

Bacterial soft rot is a disease common to many vegetables. It is a serious disease on potato, particularly in the South, and it may be responsible for heavy crop losses.

E. carotovora is a short-rod, motile, gram-negative bacterium with peritrichous flagella. This bacterium is normally found in the soil and its growth and activity there depend largely upon the supply of soil moisture. The organism can cause serious loss of seed pieces and, in waterlogged soil, can cause decay of newly formed tubers. It is also found in tubers in transit and in storage.

E. carotovora enters the host through wounds and natural openings, such as lenticels. Fresh breaks or cracks in the skin of potato tubers facilitate the entrance of this soft-rot bacterium. Bacterial soft rot is favored by rainy weather because potato tissues are then moist and the lenticels enlarge, thus providing an avenue of entrance for the bacteria. Affected tissues of tubers are typically white to cream colored, soft, and somewhat watery—especially if the decay develops in a moist atmosphere. A clear, amber liquid often exudes from the decayed part. Infected tubers break down partially or completely, and a slimy, foul-smelling rot develops, especially after frost injury. There are two phases of this rot. The slimy decay may progress into either a wet-rot stage, or it may dry up and leave lesions that are chalky white. The chalky-white type of lesion is usually found around lenticels where bacteria gained entrance earlier. In advanced stages of the disease secondary organisms usually accompany soft-rot infection and an offensive odor is present.

Control

Control bacterial soft rot by handling the potato crop carefully during harvest. Careful handling will prevent unnecessary wounds, cuts, cracks, or bruises.

Harvest potatoes in dry weather to promote rapid drying and healing of wounds and thus eliminate entry sites for the soft-rot bacterium. Avoid sunscald of the tubers.

For washing and packaging processes, use clean water only for washing and change the water frequently. Use spray jets for washing tubers. Do NOT soak tubers in deep vats for long periods of time. Dry tubers after they are washed; do not package them in tight containers. Hot-air driers hasten drying. Washed tubers may be exposed for 4 minutes to air heated to 66° C without damage. Store tubers at cool temperatures and be careful to avoid frost injury.

Exposure of tubers to solar heat for as short a period as one hour increases their susceptibility to soft rot. If the tuber temperature rises to 45° C, the tuber becomes more susceptible to soft rot through wounds. Heating or cooling tubers in water increases the amount of rot. Varieties differ in susceptibility to bruising and to infection by the soft-rot bacterium. Irish Cobbler is very susceptible to bruising and soft rot; Bliss Triumph, also susceptible to bruising, is resistant to soft rot. Kennebec and Sebago are resistant to bruising, but infected tissue becomes soft rapidly. Katahdin is resistant to invasion, but Cayuga, Teton, Irish Cobbler, and Pawnee are susceptible.

Blackleg

Causal agent: Erwinia phytophthora (Appel) Holland; *E. atroseptica (Van* Hall) Jennison sometimes indicated as causal agent.

Blackleg is a common bacterial disease of the potato. The causal organism is a short-rod, gram-negative bacterium. This bacterium is motile, with a few peritrichous flagella that act as propelling agents. In some years, this organ-

ism can cause heavy losses in potato-growing areas. It can also cause rotting of tubers in storage.

E. phytophthora has been regarded by some as a physiological race of *E. carotovora* (bacterial soft rot), *E. atroseptica* (Van Hall) Jennison, or *E. solanisapra* (Harrison) Holland; however, *E. atroseptica* is physiologically distinct from *E. carotovora,* and *E. atroseptica* only will produce typical blackleg in potato.

Characteristic field symptoms are associated with this disease. The symptoms of yellowing and severe rolling of the leaves can be recognized even when the plants are only a few inches high (fig. 17*A*). As the disease progresses, dark, inky black, and sometimes slimy lesions extend up the stems for some distance above the ground level (fig. 17*B*). These lesions can also extend downward through the stolons into the developing tubers. Under dry conditions, only the pith in the top of the plant may show blackening, and aerial tubers may form on the stems. When sufficient moisture and rather high temperatures prevail, the disease progresses rapidly and the entire plant wilts and dies.

When tubers are affected, the rot begins at the stolon end of the tuber. The lesions produced by this rot are small and dark; often only a small, circular black opening is visible on the tuber surface. In the interior of the tuber, a progressive decayed area develops into an irregular, black, soft, or slimy hollow (fig. 17*C*). Affected tubers decay rapidly in wet soil.

The blackleg bacterium may be carried in healthy-looking seed from infected plants. The seedcorn maggot, *Hylemya platura* (Meigen), and a related species, *H. florilega* (Zetterstedt) (=*trichodactyla* (Rondani)), disseminate this bacterium. These maggot flies lay eggs in the spring in the soil. The larvae, in feeding, ingest the blackleg bacterium (as well as other physiologically similar bacteria) from the soil, egg surfaces, or the contaminated surfaces of seed pieces. These bacteria are then carried through the intestinal tract of pupa and adult. Infected adults renew the cycle again in egg laying. Spread of blackleg occurs when the larvae feed on the cut surfaces of seed potatoes, making fresh wounds and, by their gnawing action in feeding, creating new invasion sites and infecting seed piece and stem. The pathogen may also overwinter in the soil and may enter the seed piece that has been freshly cut and planted under conditions unfavorable for the formation of a cork layer.

Control

The most effective measure for control of blackleg is to plant clean, whole seed. NEVER use infected tubers for seed. If cut seed pieces are used, they are less likely to be entered by soilborne bacteria if they are well suberized before being planted. Therefore, handle cut seed potatoes with care to promote rapid suberization, to avoid heating, and to prevent feeding of seed corn maggots. Plant freshly cut or properly suberized seed pieces in soil that is not too cold and wet because the cold, wet soils delay suberization and favor infection of the seed pieces.

Provide good sanitation. Destroy potato cull piles and refuse to prevent these piles and debris from becoming sources of inoculum. Practice crop rotation.

The use of antibiotics has not, in most cases, proved satisfactory or effective for control of blackleg. Antibiotics interfere with suberization and increase fungus infection of seed pieces.

Under ordinary conditions many varieties are quite resistant. Use of these resistant varieties or varieties that are not highly susceptible to the disease is recommended. If conditions are favorable for blackleg development, avoid planting the highly susceptible varieties, such as Norgold, Sebago, and Russet Sebago.

Use of nitrogen or a complete fertilizer has reduced stem infection by the blackleg. In areas where heavy fertilization is practiced, this control measure has limited value.

Brown Rot (Southern Bacterial Wilt)

Causal agent: Pseudomonas solanacearum (E. F. Sm.) E. F. Sm.

Brown rot, known also as bacterial wilt and as southern bacterial wilt, is a bacterial disease of the potato that is extremely destructive in tropical and subtropical regions. It is present

Figure 17.—Symptoms of blackleg on potato: *A*, General field symptoms; *B*, stem infection at ground level; *C*, blackleg symptoms in the tuber.

in the South Atlantic and Gulf Coast States and occurs from Maryland to Texas. It is also found in Kentucky. The disease is important in Florida and in some areas of North Carolina. It is serious in sandy, loam, clay, muck, and peat soils but is never found in marl soils. Severity of infection is increased by addition of superphosphate to the soil and is lessened by use of nitrogenous fertilizer. When temperatures are above normal in the last half of the growing season, the greatest losses from brown-rot infection occur.

The causal organism for this disease is a gram-negative, short-rod bacterium with one polar flagellum. This bacterium lives in the soil and is disseminated by infested soil. The pathogen has a wide host range, attacking plants in several genera of the Solanaceae. There are three pathogenic races of this bacterium: Races 1 and 3 attack solanaceous plants, and race 3 is highly pathogenic on potato. This bacterium has been known by various names, all of which have now been reduced to synonomy with *Pseudomonas solanacearum*. Names for this causal agent include: *Bacillus solanacearum, Bacterium solanacearum,* and *Phytomonas solanaceara.*

The first symptom of brown rot in the potato plant is a slight wilting of the leaves at the end of the branches during the hottest part of the day. Leaves of affected plants become pale green and then the leaflets take on a bronze color (fig. 18*A*). These shrivel and then die.

Affected plants recover during the cool night temperatures, but the wilting is progressive and becomes more pronounced each day until finally the plants die. The vascular bundles in the stems turn brown when they become clogged with bacteria. The clogging of the vessels in the vascular bundles causes the wilting and death of the plant by cutting off the plant's water supply. The brown color eventually appears on the outer surface of affected stems, giving them a streaked appearance. Brownish rings can be detected 1 to 2 inches above the ground level on severely affected plants. When the vascular bundles of the affected parts are cut, the bacteria, as a white, slimy ooze, soon form on the cut surface.

External symptoms on tubers may or may not be present, depending on the stage of development of the disease when the tubers are dug. The first symptom of the disease in the tuber is a brown circle, more or less complete, that can be seen in cross section slightly underneath the skin (fig. 18*B*). This circle can be readily seen around the eyes and at the stem end of affected tubers. A slimy ooze that is sticky and milky white exudes from the eyes and stem end of severely affected tubers. This exudate becomes mixed with soil, and the mixture of the bacterial exudate and soil dries and adheres to the surface of the tuber. Tubers left in the ground continue to decay; the bacteria continue to destroy the tissue that surrounds the vascular ring, and finally the skin is broken and cracks develop. Other rot organisms enter the tuber at this stage. The tubers then become a slimy mass with an offensive odor.

Figure 18.—*A*, Potato plant infected with *P. solanacearum; B,* symptoms of brown-rot infection in the tuber.

Plants with tops killed by *P. solanacearum* may bear healthy and diseased tubers. Also, plants that show no sign of the disease in their tops may sometimes produce diseased tubers.

Control

For control of brown rot, plant disease-free seed in noninfested soil. Crop rotation should be practiced, but this method of control is effective

only if solanaceous crops are excluded and the weed hosts are eliminated. Often, crop rotation as a control measure is impracticable.

Where potatoes are to be grown in sandy soil, the addition of sulfur to the soil is beneficial. Add 800 pounds of sulfur per acre to sandy soils in summer. In the fall add 3,000 pounds of dolomitic limestone per acre. Another procedure sometimes used in sandy soils is to add sufficient sulfur to lower the pH to 4.0 and follow the application of sulfur with an equivalent amount of lime.

NOTE: Read the PRECAUTIONS (inside cover page) and the recommendations for application of the particular sulfur product on the manufacturer's label. **POWDERED SULFUR IS INJURIOUS** to the eyes and respiratory passages. When you apply sulfur, wear a full-facepiece respirator.

Katahdin, Sebago, and Green Mountain are varieties that are moderately resistant to brown rot.

Ring Rot (Bacterial Ring Rot)

Causal agent: Corynebacterium sepedonicum (Spieck. & Kotth.) Skapt. & Burkh.

Ring rot or bacterial ring rot are the common names given to a highly infectious tuber disease. The causal organism is a rod bacterium that is nonmotile, short, and gram positive. Synonyms for *Corynebacterium sepedonicum* are: *Bacterium s.* Spieck. & Kotth., *Aplanobacter s.* (Spieck.) E. F. Sm., and *Phytomonas sepedonica* (Spieck. & Kotth.) Magrou. Ring rot first appeared in potato fields in the United States about 1932 and in Canada a year earlier. It now occurs in most of the potato-growing States. Because ring rot is highly infectious, the tolerance for its presence in certified seed potatoes is zero.

Symptoms associated with ring rot usually do not become evident in the plant until late in the growing season. Some plants can be infected without showing symptoms. When symptoms are present, they consist of wilting of stems, branches, and leaves. Sometimes only one or more stems in a hill may wilt and be more or less stunted, while the rest of the plant continues to look normal and healthy. Areas between veins of the lower leaves of infected stems become pale yellowish at first. The leaves of affected plants turn pale green, are mottled and chlorotic, and an upward rolling of leaf margins develops. Leaf discoloration is accompanied by a progressive wilting. Eventually the entire plant dies. When an infected stem is cut, the vascular tissues are brown. A milky-white bacterial exudate can be squeezed from the infected stem. This late-season collapse, however, is often caused by deterioration of feeding roots and is not primarily caused by the plugging of the vessels.

In tubers affected with ring rot, infection of tubers takes place at the stem end and progresses through the vascular tissue. Decay begins in a region immediately below the skin, causing a "ring-rot" type of appearance. At the stem end, when tubers are cut crosswise, infected tubers show a cheesy rot that is gray, creamy yellow or light to red brown in the area of the vascular ring. The decayed tissue is crumbly in texture (fig. 19*A*). In sufficiently advanced cases, pressure applied to the tuber will result in a separation of the tissue outside the vascular ring from the tissue on the inside of this ring. Extremely advanced stages exhibit a breakdown of the vascular ring (fig. 19*B*). Severely infected tubers, which are often invaded by other disease-causing organisms, may show external cracking and browning of the skin. Complete breakdown of infected tubers can also follow invasion of soft-rot bacterial organisms.

Ring rot is spread in diseased tubers used for seed. It is very often spread by contaminated equipment, including cutting knives, containers, and mechanical planters. Although seed may be certified as free from ring rot, a trace may go undetected and may later spread rapidly during cutting and planting operations.

Control

For control of ring rot use seed that is **ABSOLUTELY FREE** from ring-rot infection.

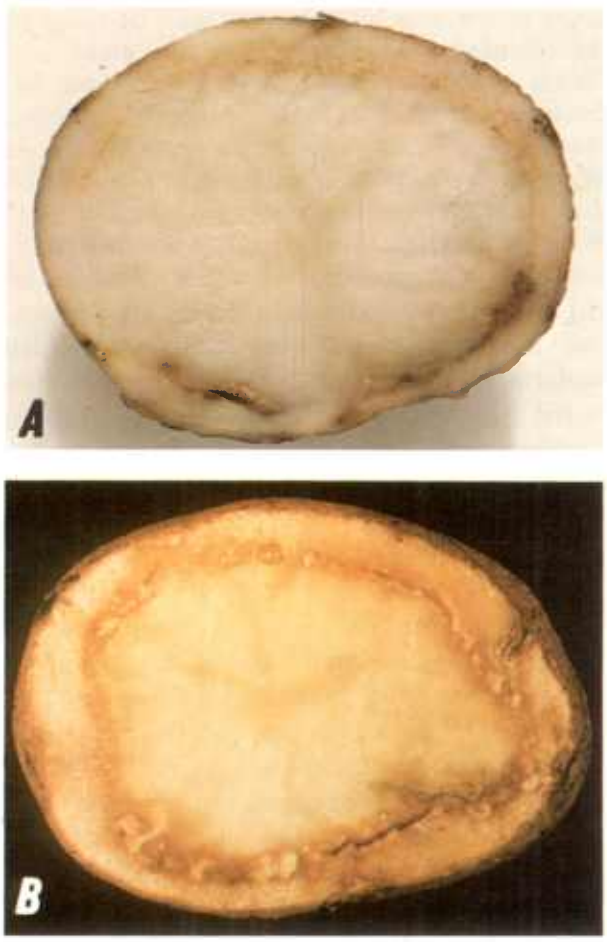

Figure 19.—*A*, Potato tuber showing early symptoms of bacterial ring rot infection; *B*, advanced stage of the infection.

Foundation or certified stock is recommended for planting.

It is extremely important to establish and maintain strict sanitation of all equipment. This includes disinfestation of cutting knives, rotary knives, containers, bags, and also disinfestation of all field equipment, such as graders, planters, diggers, and picking baskets. For disinfestation of knives, use continuously boiling water. Discard bags that are dirty or suspected of having held ring rot-infected tubers. If such bags are used, disinfest them thoroughly with formaldehyde. Disinfest machinery with a formaldehyde solution.

Before a new crop is placed in storage, if ring rot has been present, clean out the storage area completely by destroying old tubers and debris that remain in the storage area. After the storage quarters are thoroughly cleaned, disinfest the bins with formaldehyde to kill any ring rot bacteria that might still be present in the storage region.

Other control practices are (1) planting small, whole potatoes, (2) practicing crop rotation, and (3) planting resistant varieties, such as Merrimack, Saranac, and Teton.

Although *C. sepedonicum* does not overwinter in the soil, it can overwinter in infected tubers that are left in the ground. Thus, a field that has produced infected plants or tubers in the previous season should **NOT** be planted to potatoes the following year.

VIRUS DISEASES AND MYCOPLASMAS

Virus diseases of potato form one of the major disease groups affecting this crop and cause major problems to growers. Viruses cause a variety of symptoms, loss of vigor in plants, and reductions in yield.

Viruses are complex proteins. These entities are invisible without the aid of an electron microscope. Most viruses are spread from plant to plant by insects and are tuber perpetuated. Other viruses are spread by mechanical means, such as by field machinery and by the natural rubbing of plants against each other in the field. Nematodes of one genus have been known to spread at least one virus (see p. 57).

The symptoms caused by viruses that attack potato are numerous. Earlier literature refers generally to plant diseases manifesting these symptoms as "degeneration diseases" or "running out" of the crop. Virus infection can cause necrosis, such as mottling, top necrosis, and the classic necrosis of the phloem caused by leafroll virus, ring spots, and by stunting. However, plants can harbor a virus but remain symptomless.

Several diseases that were previously thought to be caused by viruses are actually caused by mycoplasmas. Mycoplasma is a term that signifies a member of the genus *Mycoplasma* and is sometimes applied to all members of the class Mollicutes. Mollicutes are minute, highly pleomorphic, plastic organisms (see Glossary, p. 75) that can be cultivated in cell-free media. These entities act as etiologic agents of virus-like diseases, and this section of the Handbook lists mycoplasmas as possible causal agents for several diseases that have generally been ascribed to viruses.

Table 1 lists the common potato diseases caused by viruses or mycoplasmas and the control recommended for each disease.

Calico

Causal agent: Strain of alfalfa mosaic virus; a clover mosaic virus.

Calico, a mosaic disease, occurs commonly in potato fields but often only on scattered plants. It causes large, irregular, yellow to cream-colored spots on the leaves, a yellow mottling or blotching of the leaflets, and a slight stunting of plant growth (fig. 20). Sometimes 70 percent of the leaf surface of affected plants may lack chlorophyll; occasionally, leaves may show only a few spots. The disease appears to be systemic because all the tubers from an infected hill will usually give rise to the same symptoms if they are used for seed. A vine and tuber necrosis have also been described as additional symptoms when the disease occurs in California.

Figure 20.—Effect of alfalfa mosaic virus, as cause of mosaic disease called "calico," on potato.

This virus is transmitted mechanically by infective sap, by aphids, and by diseased tubers.

Control

Control measures include (1) using disease-free seed, (2) roguing affected plants during the growing season, (3) eliminating volunteer alfalfa plants in or near potato fields, and (4) controlling insects.

Corky Ring Spot

Causal agent: Cause(s) unknown; suspected virus.

Corky ring spot is a disease known in European countries, Indonesia, and South Africa. It was found in Florida in 1946 but has not been reported from any other Southern State. Potato diseases in the literature as "sprain," "spraing," "internal rust spot", and "potato stem mottle" may be, in part, synonyms for corky ring spot.

Plants from tubers infected with corky ring spot are sometimes dwarfed with leaves that are malformed and mottled. Some leaves may show an irregular necrotic spotting. No fungus or bacterial growth is evident in the dead tissues.

Tubers affected with corky ring spot become irregular in shape during the early stages of growth because of the development of deep cracks and shallow corky depressions on the tuber surface (fig. 21*A*). Brown, concentric rings develop on many of the tubers. The tissue cracks within these arc-shaped lesions. In the flesh of the tuber, rusty-brown lesions of irregular shape and size may appear (fig. 21*B*). The discolored flesh has a corky texture.

The pathogen causing corky ring spot is soilborne and tuber perpetuated. The tobacco rattle virus, transmitted by nematodes in the genus *Trichodorus*, has recently been associated with corky ring spot. In tests in the Netherlands, where the tobacco rattle virus was compared with corky ring spot, stem-mottle, and spraing isolates, the tobacco rattle virus alone caused stem symptoms and tuber necrosis on Eigenheimer variety after root inoculation. The stem mottle isolate caused heavy mottling on

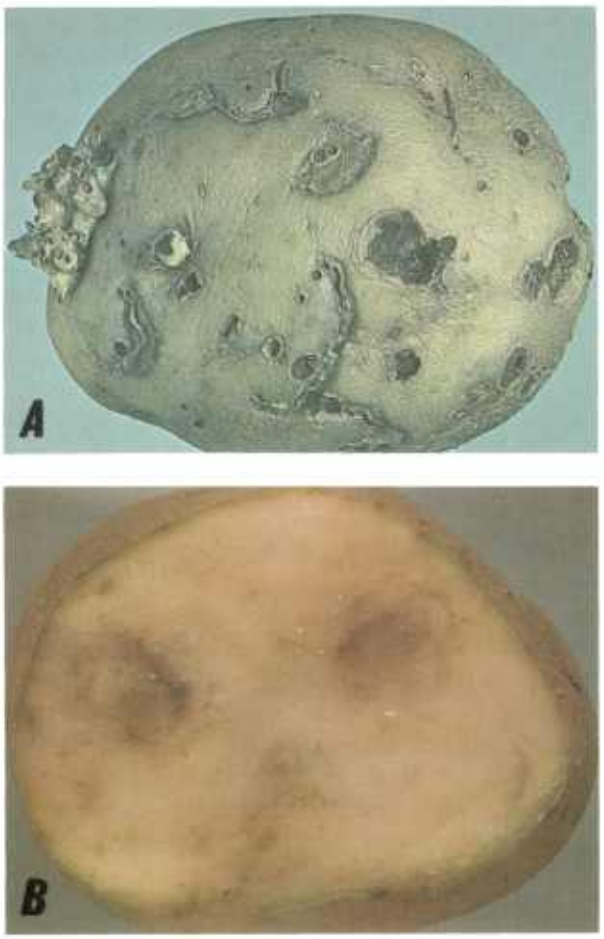

Figure 21.—Corky ring spot symptoms: *A*, On tuber surface; *B*, in tuber flesh.

the stems and severe tuber deformation and necrosis after leaf inoculation.

A similar soilborne disease, termed mop top, has been described in Europe. This latter disease is transmitted by *Spongospora subterranea* (Wallr.) Lagerh., the soil fungus that causes powdery scab.

Control

No control for corky ring spot is known. Recommendations are to use disease-free seed and avoid planting in infested soil.

Haywire (Green Dwarf Disease)

Causal agent: Sugar beet curly-top virus.

Haywire (green dwarf disease), a virus disease often found in mountainous States, is more serious in high valleys and on high plateaus. It does not seem to increase rapidly, and sometimes only a few scattered plants in the field are affected. The virus is transmitted by the beet leafhopper, *Circulifer tenellus* (Baker).

The symptoms on haywire-affected plants often resemble those caused by leafroll virus, psyllid yellows, and Rhizoctonia canker. The disease is characterized by late emergence or missing hills and by extreme dwarfing. Sometimes dormant seedpieces produce sprout tubers. Affected plants that do emerge are severely dwarfed. They have a rosette appearance because of a cessation of the terminal growth, a shortening of the internodes, and an increase in the number and development of axillary shoots. Often the terminal growth is malformed; a cluster of small leaves pinched together forms the growing point. At midseason a curling and yellowing of the top leaves occur. The leaflets also are usually rugose, erect, stiff, rolled, and pointed. Often these leaflets are purple at the tips and margins. Petioles and stems may also show swellings at the nodes with a red or purple pigmentation. Sometimes aerial tubers are formed in the leaf axils.

If a plant is affected with haywire when it emerges from the soil, it will produce only very small tubers that weigh less than one-half ounce, and the tubers will be set close to the stem at the soil line. When symptoms appear at midseason, 1- to 2-ounce tubers sometimes develop. These tubers are likely to show symptoms of phloem (net) necrosis and they seldom sprout.

Control

Control measures are to (1) use disease-free seed, (2) rogue affected plants from the fields as soon as they are noticed, and (3) control insect vector and weeds.

Interveinal Mosaic (Supermild Mosaic or Paracrinkle)

Causal agents: Interaction of viruses X, S, and M.

A disease condition in the potato, called interveinal mosaic, was identified in the Irish Cobbler potato variety. Similarly, a disease called "leafrolling mosaic" was identified in

Green Mountain variety. Other workers have called this disease complex supermild mosaic and paracrinkle.

The symptoms caused by interveinal mosaic in potato are a slight dwarfing, accompanied by diffuse mottling (fig. 22), wrinkling, slight ruffling, and an upward rolling of the upper leaves. In hot weather, these symptoms are usually masked or are very mild.

The cause of these symptoms in potato results from the interaction of potato viruses X, S, and M. Virus M is called the IVM factor in this complex. These viruses can be present in the potato without producing symptoms. They can be transmitted by infective sap and their presence in the plants can be detected by indexing diseased material on suitable indicator hosts or by studying their serological reactions with appropriate antisera.

Control

This disease complex can be controlled in some varieties by clonal selection of virus-free stock. Selections can be made by using indicator-plant testing or serological determinations. The variety Saco is resistant to potato virus S.

Potato virus M can be transmitted by the green peach aphid, *Myzus persicae* Sulzer. Therefore, control aphid vectors. In all growth and handling phases, practice good sanitation.

Latent Mosaic (Potato Virus X, Potato Mottle Virus)

Causal agent: Potato virus X.

The latent mosaic in potato is caused by infection of the plant with potato virus X, commonly called PVX.

PVX usually produces no symptoms on most potato varieties, particularly those in cultivation for years previous to 1920. This symptomless characteristic causes the PVX virus to bear the pseudonym "healthy" potato virus.

Some potato varieties affected with PVX will show a mild leaf mottle (fig. 23) called weather mottling by potato growers and inspectors. This symptom is noticed particularly after several days of cloudy weather. It disappears or is masked under high-light intensity, such as in bright sunlight. Affected plants may be stunted and may have necrotic spots on the leaves. Sometimes spots may occur in the tubers. PVX was long considered to be harmless.

Some European varieties, such as Arran Crest, Epicure, and King Edward, develop an acute necrosis of the growing tips. This type of infection is systemic and usually results in the

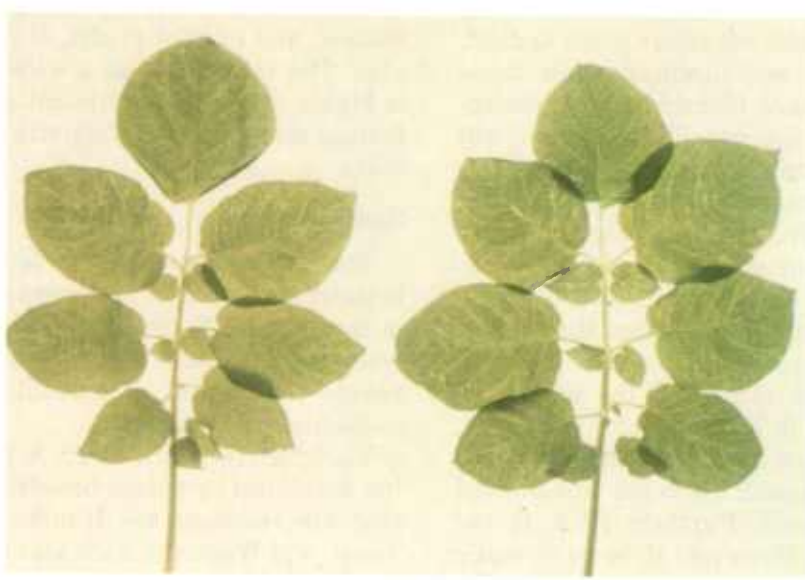

Figure 22.—Leaf symptoms caused by interveinal mosaic.

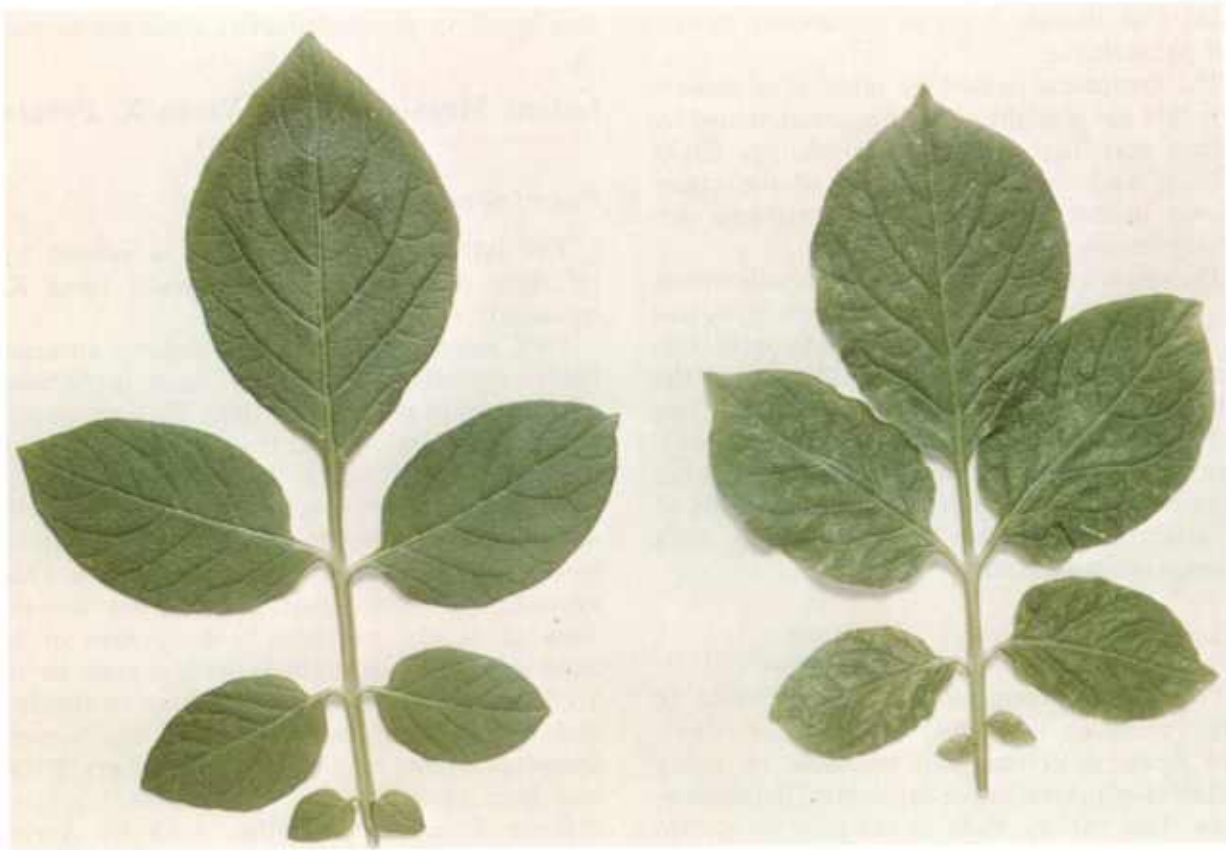

Figure 23.—Potato leaves showing symptoms of potato virus X.

death of infected plants, the dying beginning at the top of the main stem and then proceeding down from the tip until the entire plant is dead. Thus, the virus is self-eliminating in these varieties. If tubers are formed from these infected plants, they are usually small and will often show an internal necrosis.

American varieties, such as Green Mountain, Irish Cobbler, and Russet Burbank, and other old varieties are invariably infected with PVX. While the virus was long considered to be harmless, experiments have shown that it can reduce yield considerably. Losses are influenced by the strain of the virus and the particular potato variety that is infected.

Most new varieties are free from the virus for a few years because PVX is not transmitted through the true seed. Further, PVX is not insect transmitted. However, it is very easily transmitted mechanically by sap inoculation. PVX is also transmitted by the cutting knife, by cultivating and spraying equipment, by leaf contact, and by root grafts. It is tuber perpetuated. The virus also has a wide host range and is highly resistant to physical agents. All these factors maintain its infectivity and transmissibility.

Control

The first recommendation for control of PVX is strict sanitation. The second recommendation is to plant PVX-free foundation seed stock if possible. Cultivate when plants are small to reduce the hazard of spreading the virus by contaminated equipment.

Varieties resistant to PVX infection are being developed by potato breeders. New varieties that are resistant are Hunter, Reliance, Saco, Tawa, and Wauseon. Saco also has resistance to viruses A and S.

Leafroll

Cause: Leafroll virus.

Leafroll is probably the most serious virus disease of potato. It is a disease of great economic importance because infection of plants with this virus causes a marked reduction in yield. Yields from affected plants may be reduced by one-third or one-half of normal. Tubers of leafroll-infected plants are small to medium and shaped normally. The virus is present in many of the presently grown varieties. At least five strains of leafroll have been reported in potatoes.

As the name implies, leafrolling is the main symptom of the disease. In most varieties, the first symptoms become noticeable about a month after the plants appear above the ground. The leaflets of the lower leaves roll up at the edges and become somewhat papery, brittle, and leathery to the touch. Affected leaves also rattle if brushed with the hand. As the plant grows, the rolling appears on the higher leaves and eventually affects the whole plant (fig. 24). The rolled leaves are lighter in color than healthy ones. In some varieties, a reddish or purple discoloration occurs on the underside of the leaves. Often, the plants will be dwarfed and rigid.

Early current-season infection produces a characteristic rolling of the upper leaves but no dwarfing. Plants infected late in the growing season often remain symptomless. However, tubers of susceptible varieties will develop phloem necrosis either before or after harvest. Visually, this necrosis is a network of brown to dark brown strands of dead tissue that extends throughout the stem end of the tubers (fig. 25). This condition is also called net necrosis, and it appears in tubers of plants that become infected in the field. It is rarely, if ever, derived from the seed tubers.

Leafroll virus is transmitted primarily by the green peach aphid (*Myzus persicae* Sulzer); however, other species of aphids occasionally serve as vectors. The virus is not mechanically transmitted by sap inoculation but is tuber perpetuated. If the virus is spread by aphids late in the season, newly infected plants do not show typical symptoms. Tubers from these late-infected plants, if used for seed, will produce diseased plants.

In northern New England, phloem (net)

Figure 24.—Potato plant infected with leafroll virus.

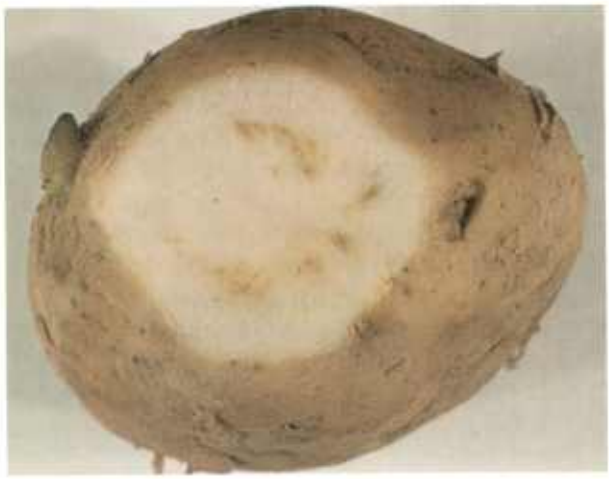

Figure 25.—Phloem necrosis (net necrosis) in tuber following current-season infection with leafroll virus.

necrosis is rarely evident at harvesttime, but it frequently develops in susceptible varieties in storage from infection that took place in the field during the previous growing season. Phloem (net) necrosis is frequently present, however, at harvesttime in Washington in tubers from Russet Burbank (Netted Gem) plants infected in the current season. This condition develops further in this variety in storage. Phloem (net) necrosis has also been known to develop in Russet Burbank tubers from chronically infected leafroll plants.

Control

To control leafroll satisfactorily: (1) Select and use disease-free seed; (2) isolate the seed plots; (3) rogue diseased plants; (4) control aphid vectors; (5) harvest seed plots early; and (6) grow resistant varieties.

Katahdin and Sequoia are field resistant to leafroll infection. Penobscot is a new variety that has resistance to leafroll. Varieties that are very susceptible to natural infection in the field are Chippewa, Green Mountain, and Russet Burbank.

Phloem (net) necrosis is controlled primarily by use of resistant varieties. Varieties that are resistant to the leafroll-phloem necrosis complex are: Alamo, Cherokee, Chieftain, Chippewa, Houma, Katahdin, Merrimack, Pungo, Redskin, Saco, Sebago, Reliance, and Wauseon. Avoid planting the highly susceptible varieties, such as Green Mountain, Irish Cobbler, and Russet Burbank. If susceptible varieties are grown, market them at harvesttime before an excessive amount of phloem (net) necrosis develops. If potatoes are to be stored, store them for 60 days at 3° C (35° F) or 21° (70°). Because of the relatively high soil temperatures that precede harvest in southern New England and on Long Island, N.Y., phloem (net) necrosis is not usually a problem in those areas.

Aphid vectors should be controlled to prevent spread of the leafroll virus.

Mild Mosaic

Causal agent: Potato virus A; strains of virus A.

Virus A is a well-known and extremely prevalent virus disease of potato. It is a disease common in all potato-growing areas, and it is the most difficult of all potato diseases to recognize under conditions of bright sunlight and high temperatures during the growing season. Detection of this disease is almost impossible when prolonged periods of dry weather with high temperatures occur, because the field symptoms are so inconspicuous.

The disease results from the action of one or another of the strains of the mild mosaic virus. During early potato research, many scientists believed that mild mosaic resulted from a combined synergistic effect of potato virus A and potato virus X (PVX). These two viruses can and often do react synergistically, particularly in the case of symptomless carriers of PVX. Virus A alone is capable of producing mild mosaic in some potato varieties.

The characteristic symptom of mild mosaic infection is a chlorotic mottling in which yellowish or light-colored areas alternate with similar areas of normal green in the leaf (fig. 26). The mottled areas vary in size and are not restricted by the leaf veins. A slight crinkling is usually present and, under conditions favorable for disease development, the margins of the leaflets may become wavy or ruffled. The severity of symptom expression is influenced by the presence or absence of PVX and by the particular strains of viruses A and X involved. Three strains of virus A have been recognized.

Diseased plants tend to be slightly erect at

Figure 26.—Leaflet of Green Mountain potato plant infected with mild mosaic virus. Note the yellow or light-green patches interspersed in the normal green.

first and are stunted; then they droop and die prematurely. Tubers produced from virus-A infected plants are smaller than healthy tubers. In some European varieties very sensitive to virus A, the virus produces a top necrosis in the plant. Sometimes necrotic tubers are produced as well, as in the variety British Queen.

Virus A is tuber perpetuated and is aphid transmitted. It is not readily sap transmitted.

Control

To control mild mosaic: (1) Plant disease-free seed potatoes; (2) practice rigid control of insects; (3) rogue diseased plants carefully and thoroughly; and (4) kill vines in seed plots early.

Many varieties are field resistant. These include: Alamo, Cherokee, Chieftain, Chippewa, Houma, Katahdin, Kennebec, Merrimack, Mohawk, Monona, Ona, Penobscot, Pungo, Redskin, Sebago, and Wauseon. Although many varieties are field resistant to mild mosaic, they are not immune because virus A can be transmitted to them experimentally. However, for all practical purposes, they can be considered to be resistant to virus A.

For detecting potato virus A in diseased material, when potato virus X is present, Kohler's A6 [*Solanum demissum* Lindl. x Aquila hybrid A6] is useful as a local-lesion indicator.

Purple-Top Wilt and Related Diseases

Causal agent(s): Aster yellows "virus"; mycoplasma suspected.

Purple-top wilt, one of a group of diseases that include purple top, blue stem, bunch top, purple dwarf, late-breaking virus, and possibly yellow top, is a disease found in the northern and eastern parts of the United States. It has been found from New York State westward to Minnesota and North Dakota and southward through the mountainous sections of Pennsylvania and West Virginia. It has not been found in the South. Purple dwarf and late-breaking virus have been found in Oregon. Purple-top wilt is a disease transmitted by the aster leafhopper, *Macrosteles fascifrons* (Stål), and possibly by other species of leafhopper.

The first symptoms of purple-top wilt occur at the apex of the plant. The young leaves fail to enlarge normally, and the leaflets roll upward. Pronounced rolling occurs at the base of the leaflet.

In varieties that normally have a red pigment, a reddish-purple color appears on the foliage and stems of diseased plants. The color is most intense at the base of the leaflet where the curling is also most evident. In varieties that lack pigment, the topmost leaves become chlorotic and a light-green or yellow cast often appears. An abnormal number of axillary shoots develop, which also show a yellowish cast. The axillary shoots become swollen at the base and often form distinct aerial tubers (fig. 27).

In stems, the vascular tissue turns brown at the same time that the foliage symptoms appear. The abnormal color may extend only a few

Figure 27.—Symptoms of purple-top wilt in potato shoots. Note the swollen stems of the axillary shoots.

inches into aboveground stems, but it usually extends well into the stolons. Necrotic flecks are often present in the pith at the lower part of the stem. Affected plants generally wilt within 2 weeks after the symptoms appear. Death of the plant then occurs. Plants affected in late summer mature late and, when these plants are killed by frost, the stems turn intensely black.

Tubers from infected plants are sometimes soft and spongy. These tubers may also develop a stem-end necrosis. Sometimes they fail to germinate or may develop spindling sprouts.

In advanced stages of purple-top wilt, the aboveground symptoms resemble those caused by psyllid yellows, but the tuber symptoms do not.

Control

Inasmuch as purple-top wilt and related disease situations are caused by various strains of the aster yellows "virus," the most important control measure for purple-top wilt in potato is the use of an effective insecticide to eliminate the leafhopper vectors, one of which is the aster leafhopper. Other recommended practices include (1) planting healthy seed potatoes, (2) roguing diseased plants, and (3) controlling weeds that may act as reservoir hosts for the virus.

Rugose Mosaic (Potato Virus Y)

Causal agent: Potato virus Y; potato virus Y plus potato virus X.

Rugose mosaic is a serious disease of potato. It is important because it affects the seedstocks, causing a degeneration or "running out" of these stocks.

Plants emerging from tubers affected with rugose mosaic are dwarfed and the leaves are mottled. The leaf-pattern symptom is a distinct mosaic and differs from that caused by mild mosaic in that the discolored areas are more numerous, smaller, and are distributed closer to the main veins. Leaf mottling may be readily masked at high temperatures, but the disease can be identified by the crinkling and rugose appearance of the surface and margins of the leaves (fig. 28*A*). Veins on the underside of lower leaves often show necrotic areas resembling black lines drawn with a pencil. Affected plants are stunted and die prematurely (fig. 28*B*).

Symptoms caused by current-season infection differ from those that develop in plants grown from tubers infected with rugose mosaic during the previous season. Current-season symptoms are characterized by a burning and discoloration of leaf veins and blades, brittleness, leaf

dropping, and premature death of the plant. The necrosis starts as spots on the leaflets and progresses until the leaflets and the entire leaf die yet remain clinging to the stem. Sometimes these symptoms appear only on a single shoot in a hill, but as the season progresses other shoots in the same hill may show similar symptoms. If infection occurs late in the season, plant symptoms may not appear but the tubers from these infected plants will carry the disease. The only tuber symptom is a marked reduction in size caused by damage to the plant top.

Figure 28.—Rugose mosaic: *A*. Crinkling and rugose appearance of affected leaf (right) as compared to healthy leaf (left); *B*, symptoms of rugose mosaic on potato plant.

Potato virus Y (PVY) appears to be the primary cause of rugose mosaic. The presence or absence of potato virus X (PVX) also influences symptom expression. A difference of opinion exists as to whether rugose mosaic is caused by PVY alone or by the combined action of PVY and PVX. However, rugose symptoms in certain varieties can be produced by PVY alone.

PVY is transmitted mechanically by infectious sap. It is also transmitted readily by the green peach aphid, *Myzus persicae* Sulzer, without requiring an incubation period in the body of the insect.

Control

To control rugose mosaic (1) plant disease-free seed potatoes, (2) enforce rigid control of the insect vector, and (3) carefully rogue diseased plants during the growing season. Roguing should start when the plants are 5 to 6 inches high and should be repeated every 7 to 10 days until midseason. There is no varietal immunity to the disease, but Chippewa, Katahdin, Kennebec, Monona, and Snowflake varieties are resistant.

Spindle Tuber

Causal agent: Potato spindle tuber viroid; also the unmottled curly dwarf strain of spindle tuber.

An unusual disease condition of potato in the United States and Canada is caused by the potato spindle tuber viroid. Its presence can cause a reduction in yield, and when attacks are severe, it can also cause a reduction in tuber grade.

The causal agent for this disease is a very unusual entity. It is a minuscule particle that appears to exist in the plant as free ribonucleic acid. The small, infective molecule that causes

potato spindle tuber disease has a molecular weight of about 80,000 daltons. It is the second smallest known viral ribonucleic acid of an independently replicating virus.

Potato spindle tuber viroid-infected plants are extremely difficult to detect. The symptoms on potato plants are more obscure than the more obvious tuber symptoms. The plants tend to be more erect than when noninfected, and infected plants are spindly in growth. A narrowness of the shoots and a dwarfing occur, and the leaves are darker green than normal (fig. 29*A*). The leaflets tend to be twisted and are subtended from the main stem at about a 45° angle. If an observer stands above the plant and looks directly down into its crown, the plant will appear to twist slightly in a clockwise direction. These symptoms are most prominent at high temperatures and can be considerably masked when temperatures are low.

The most obvious symptom of this disease is the elongation of the tuber. The tubers become long, are more or less cylindrical, and have tapered ends. The eyes of the viroid-infected tubers are numerous and very conspicuous, much more so than in healthy tubers (fig. 29*B*). A distinct "eyebrowing" of the tuber flesh occurs over each eye.

Unmottled curly dwarf has been considered a strain of the potato spindle tuber disease. Plants infected with this strain exhibit foliar symptoms similar to those found on plants infected with potato spindle tuber but show a more pronounced curling of the leaflets. Tubers from unmottled curly dwarf-infected plants are more nearly round, less elongated, and rougher than tubers from spindle tuber-infected plants. Frequently, the tubers infected with the strain exhibit deep growth cracks and sometimes develop internal necrosis.

The potato spindle tuber viroid is very readily sap transmissible. It can be transmitted to a wide range of host plants. In many of these plants the viroid remains symptomless, but *Lycopersicon esculentum* Mill. cv. Rutgers (tomato) is a good systemic indicator host for this viroid. In 1971, *Scopolia sinensis* L. was reported to be a local-lesion host for spindle tuber. Sprouts and eyes from healthy potatoes can be infected with sap from freshly cut, infected seed pieces. The viroid can also be transmitted by grafting, by cutting knives, by contaminated equipment, and by many insects. The grasshopper (*Melanoplus* spp. and possibly other species), flea beetle (*Epitrix cucumeris* (Harris)), tarnished plant bug (*Lygus lineolaris* (Palisot de Beauvois), and the larvae of the Colorado potato beetle (*Leptinotarsa sanguinolenta* (Provancher)) have been reported as vectors.

Control

Spindle tuber is a disease that is difficult to control. Use foundation seed potatoes, free from the disease, whenever necessary. Grade seed potatoes before sprouting and dry the cut seed pieces before they are planted. Other recommended practices are to (1) use tuber-unit seed plots, (2) rogue diseased plants early in the season, (3) disinfest all field equipment, (4) complete cultivation and hilling of the plants while the plants are small, and (5) control insect vectors.

Stem-End Browning

Causal agent: Cause unknown; virus suspected.

Stem-end browning is a condition that develops in stored potatoes in certain susceptible varieties, such as Irish Cobbler and Green Mountain. Seasonal conditions, cultural factors, use of fertilizers that have a high chlorine content, and adverse storage temperatures have some effect on the prevalence of stem-end browning. The condition is often confused with net necrosis caused by the leafroll virus.

In tubers affected by stem-end browning, the vascular ring is discolored. The discoloration in both the xylem and the phloem elements is darker than the discoloration that derives from phloem (net) necrosis. The extent of the discoloration is shallow and is limited to about one-half inch from the stem end of the tuber. The discoloration caused by phloem (net) necrosis is usually deeper.

Control

The most practical control measure for stem-end browning is to plant resistant varieties, two of which are Katahdin and Chippewa. Proper storage temperatures are also recommended.

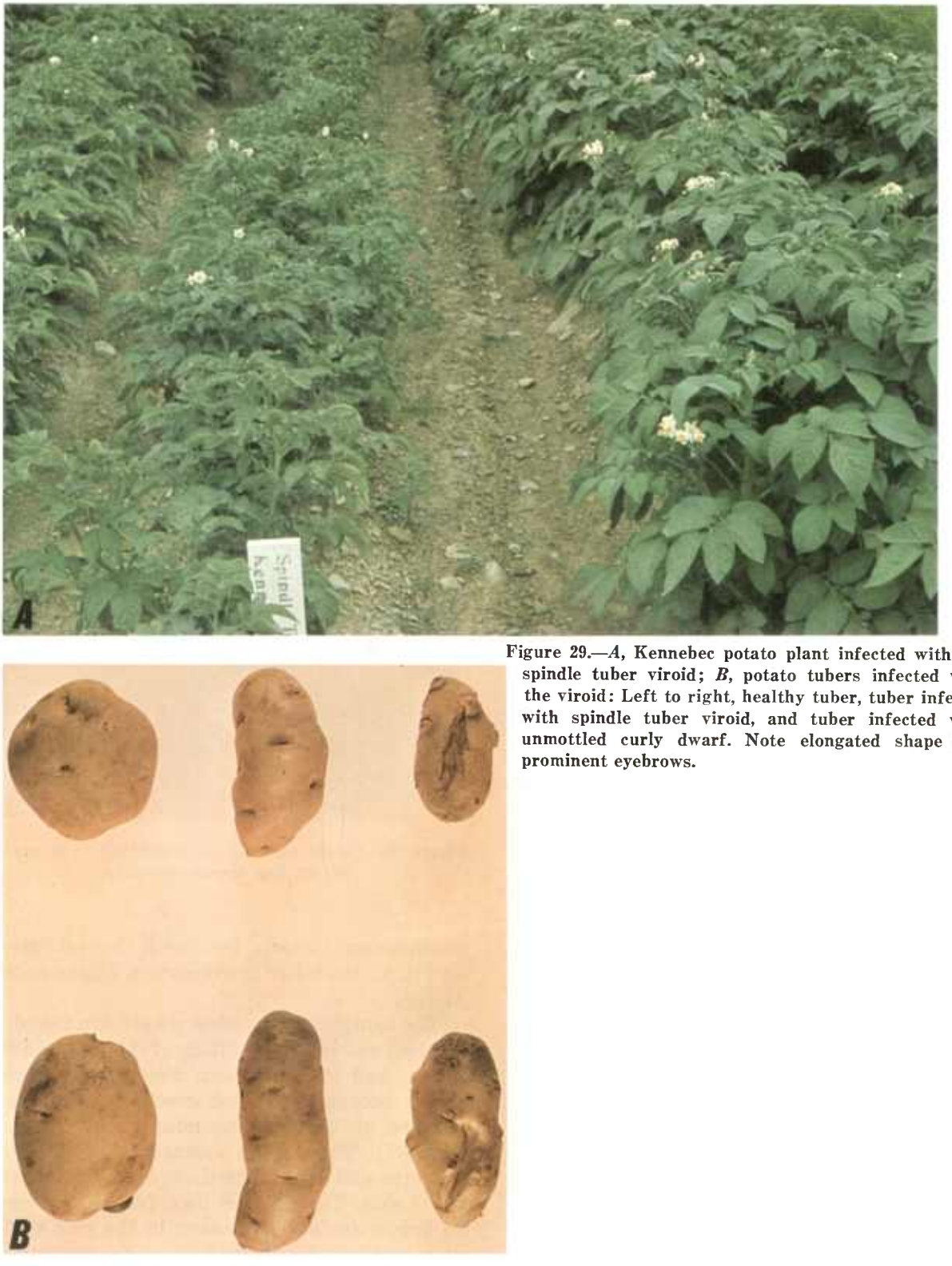

Figure 29.—*A*, Kennebec potato plant infected with the spindle tuber viroid; *B*, potato tubers infected with the viroid: Left to right, healthy tuber, tuber infected with spindle tuber viroid, and tuber infected with unmottled curly dwarf. Note elongated shape and prominent eyebrows.

Witches' Broom

Causal agent: Virus suspected; possibly a mycoplasma.

Witches' broom is an uncommon disease that occurs primarily in the Pacific Northwest.

Plants that are affected by witches' broom are dwarfed and chlorotic. Almost all of the eyes of the seed tuber will produce shoots, and these shoots form side shoots, so that in the end a diseased plant with many slender, spindly, cylindrical stems is produced. The leaf nodes are larger than normal. Usually the leaves are simple, small, and velvety in texture. Infected plants often do not grow taller than 9 inches (fig. 30). In plants affected with witches' broom, flower and seedball formation is usually greater than in healthy plants. Aerial tubers sometimes form and produce leaf growth or flower parts at the eyes.

Affected plants produce many marble-sized tubers. Frequently, these tiny tubers are borne in chains along the stolons that grow out from the eyes. The small tubers often sprout the same season and produce large numbers of spindling sprouts.

This disease is tuber perpetuated. It is not sap transmissible. It can be transmitted by graft and by dodder (*Cuscuta* spp.). In Japan, *Scleroracus flavopicta* (Ishihara), a leafhopper, has been reported as a vector of the disease. In western Canada two species of leafhoppers in the same genus, *S. dasidus* Medler and *S. balli* Medler, have transmitted the disease from clover (genus *Trifolium*) and alfalfa (*Medicago sativa* L.) to potato and from potato to clover, but not from potato to potato.

Three strains of the disease have been identified in British Columbia, one of which appears to be related to tomato big bud.

Control

For control of witches' broom, plant disease-free seed and practice careful roguing to control this disease.

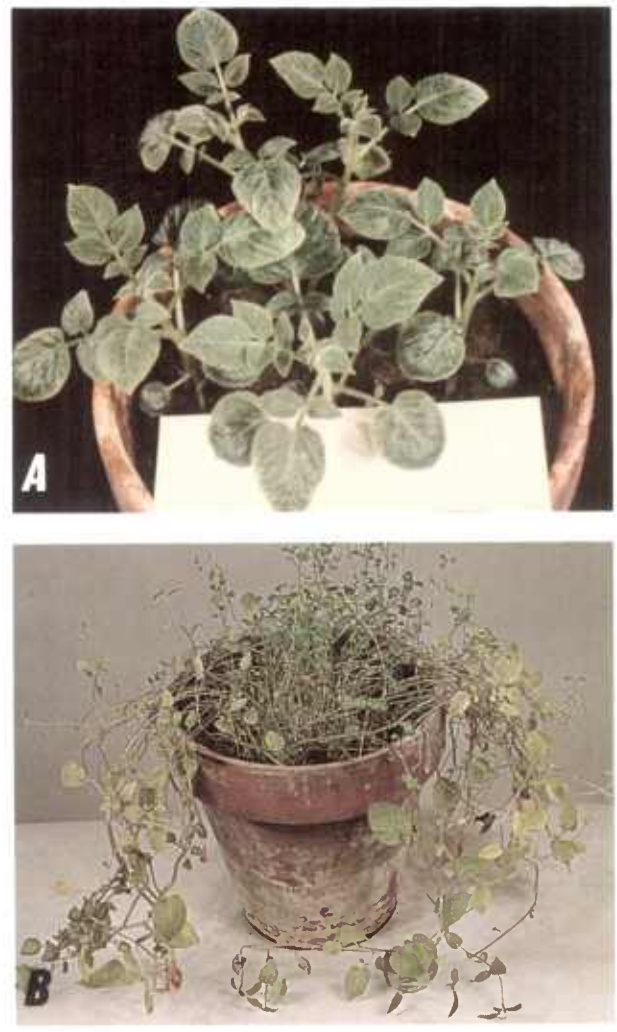

Figure 30.—Potato plant showing mild (*A*) and severe (*B*) witches' broom symptoms.

Yellow Dwarf

Causal agent: Potato yellow dwarf virus.

Yellow dwarf is a serious disease of potatoes in New York State. It also causes losses in southeastern Canada, the North Central States, and in all the other Northeastern States except Maine.

The symptoms of yellow dwarf are found in the leaf and in the condition of the plant, in the stems, and in the tubers. Foliage of affected plants becomes yellowish green, and the upper surface of the leaf becomes slightly rugose (fig. 31). There is an upward roll to the leaf margins and a downward curve of the longitudinal axis. The plant is dwarfed. Rust-colored to brown spots are common in the pith of the stem and in the cortex of the nodes. Sometimes these spots are found in the internodes. They

Figure 31.—Potato plant showing typical yellow dwarf symptoms.

appear shortly after yellowing of the foliage and eventually can extend the entire length of the main stem. Dying of the plant from the tip downward is characteristic, but sometimes this symptom is lacking.

Tuber symptoms vary. Infected plants often produce small, misshapen tubers. When tubers are cut in cross section, small, necrotic areas are found scattered throughout the flesh. Growth cracks are common. In warm soil, seed pieces from infected tubers often fail to germinate. When seed pieces do produce shoots, the shoots die before they reach the surface of the soil.

The yellow-dwarf virus overwinters not only in infected potato tubers but also in clover plants, chrysanthemums, and viruliferous clover leafhoppers. The disease is transmitted by several species of clover leafhoppers, including *Agallia quadripunctata* (Provancher) and related species. It can also be transmitted, with difficulty, by sap inoculation. At least three strains of the virus are recognized by their reaction on indicator hosts and by vector specificity.

Control

For control of yellow dwarf (1) plant disease-free seed potatoes in fields isolated from clover, (2) rogue diseased plants, and (3) control insect vectors. The newer and more effective insecticides have played a major role in the effective control of this disease. The Sebago variety is field resistant.

NEMATODES

Nematodes are tiny worms that live in soil and water. Some genera and species of these organisms are parasitic on potato roots or tubers or both. Nematodes are capable of causing mild to serious disorders. The severity of disease development depends on the nematode species, nematode population size, and the susceptibility of the host.

Nematodes have been implicated in the spread of other disease organisms. One species, *Trichodorus christei* Allen, is reported to transmit the corky ring spot virus. The root-knot nematodes (*Meloidogyne* spp., notably *M. hapla* Chitwood and *M. incognita* Chitwood) has been implicated in increasing the severity of *Pseudomonas solanacearum,* a bacterial disease of the potato (see p. 40). An interaction between *Pratylenchus penetrans* (Cobb) Filipjev & Schuurmans Stekhoven and the incidence and severity of *Verticillium albo-atrum* Reinke & Berth., a fungus disease of potato causing Verticillium wilt, has been reported. Thus, it is probable that nematodes may act synergistically with other pathogens in the development of potato disease complexes. Table 1 lists the common potato diseases caused by nematodes, the specific agent for each disease, and the recommended control.

Golden Nematode

Causal agent: Heterodera rostochiensis Woll.

The golden nematode, sometimes called the potato root eelworm, is a very serious parasite on potatoes. It occurs in areas of northern Europe. In South America, in the Andes Mountains of Peru, the presence of this nematode is believed to be endemic. Fortunately, the distribution of the golden nematode has been limited in the United States. It was found on Long Island, N.Y., in 1941. By careful and strict quarantine regulation, it was contained in this

area until 1968, when it was found in Steuben County in western New York State.

The name "golden nematode" is derived from the golden color of the cysts. These minute cysts are pinhead in size and contain many eggs. The cysts persist in the soil and can be transported from one location to another in infested soil. Infested soil may cling to the surface of tubers or may be found on equipment, in containers, or on the roots of other plants. The eggs from these cysts hatch to form larvae, which soon attack host plants for survival. These larvae enter roots, stolons, or tubers and align themselves near the vascular system. From this position, the female larva swells and finally breaks through to the outside surface of the host as a generally transparent but sometimes opaque body. This body undergoes color changes from golden to reddish brown. The female dies after developing several hundred eggs. The cysts, or dead female bodies containing eggs and larvae, are the diagnostic element for defining infestation with this nematode. The male remains wormlike in shape.

The aboveground field symptoms caused by this nematode include chlorosis, some stunting, wilting of the plant during the heat of the day, and early death of the plants. On the whole, plant growth is poor. As the outer leaves of plants die back, the plants become tufted at the top.

Because a golden-nematode infestation interferes with normal root action and because attacked plants have a less than normal growth period, the yield and size of the tubers are often reduced. Yield loss, however, can depend on many factors besides level of infestation. The season and the type of soil both have an effect because infestations are worse in dry weather and worse on light soil than on heavier soil.

Races (sometimes called biotypes) of the golden nematode exist, and breeders are attempting to breed potato varieties resistant to this nematode. Resistance factors exist in the wild Argentine potato, *Solanum vernei* Bitt. & Wittm. ex Engl., and in several clones of the cultivated Andean potato, *S. tuberosum* L. ssp. *andigena* (Juz. & Buk.) Hawkes. By crossbreeding material from the *andigena* crosses with that from *S. multidissectum* Hawkes, resistance to the A and B biotypes of the golden nematode has been found. Other *Solanum* species that are resistant are *S. kurtzianum* Bitt. & Wittm. ex Engl. and *S. velascanum* Bitt. & Wittm. (syn.: *S. famatinae* Bitt. & Wittm.).

Control

To control this nematode, observe strict sanitation and use good cultural practices. **DO NOT** move soil or nursery stock from infested areas to noninfested areas. The enforcement of **strict** plant quarantine regulations is vitally important.

In some places, long crop rotations are important for control. When infestation is severe, exclude potatoes and tomatoes from the land for upwards of 8 years or more. The soil should be fumigated with DD or Vorlex. Contaminated equipment should be disinfested with steam.

In infested areas, plant certified nematode-free seed and use varieties resistant to this pest. Varieties with golden-nematode resistance are Peconic and Wauseon.

Lesion Nematode

Causal agent: Pratylenchus spp., mainly *P. penetrans* (Cobb) Filipjev & Schuurmans Stekhoven, and *P. pratensis* (DeMan) Filip.; and many other reported species.

Lesion nematodes have a wide host range. These two species have been found on potato roots. Where infestations are severe, yield can be reduced.

On potato tubers pimples appear that later change to black depressions. When an infestation is severe and the pimples and depressions are numerous, tubers are disfigured and become unmarketable.

A synergistic action between *Verticillium albo-atrum* (Verticillium wilt) and *P. penetrans* in the incidence and severity of Verticillium wilt has been demonstrated. It is probable, therefore, that these nematodes may also act synergistically with other pathogens in the development of potato disease complexes.

Control

Control lesion nematodes by fumigating the soil. Chloropicrin and 1,3–D, applied in the fall,

have been used to control lesion nematodes. DD and DD-Mencs have also been used, and EDB has been approved for potatoes.

Additional practices to help control this nematode are to (1) provide good sanitation, (2) practice crop rotation, and (3) use nematode-free seed.

Potato-Rot Nematode

Causal agent: Ditylenchus destructor Thorne

The potato-rot nematode is a serious pest of the potato in Europe. In North America this nematode was first found parasitizing potatoes in Prince Edward Island, Canada. It has also been found on potatoes in Idaho and Wisconsin.

In the United States no plant symptoms have been associated with the attack of this pest. However, in England brown stem lesions and distorted leaf bases and stems occur.

The first symptoms on tubers are the appearance of small, grayish patches or discolored spots or holes. Only the superficial tissue is involved and the spots resemble the injury caused by wireworms. This tissue shows a whitish, glistening area where cells have become loosened from one another. Colonies of nematodes are present in these lesions. In later stages of infestation a grayish or brownish granular decay begins. This is followed by a drying, shrinking, and cracking of the surface. When areas coalesce, the surface may become soaked but it remains soft to the touch. The decay is easily confused with that caused by Fusarium dry rot; the cracking resembles symptoms of bacterial ring rot.

The long, slender nematodes that cause the decay are most prevalent in the sound tuber flesh around the decayed tissue. Saprophytic nematodes that lack stylets frequently invade and feed on decaying potato tissue.

Control

To control the potato-rot nematode use noninfested tubers for seed. Practice strict sanitation. Do not plant potatoes in infested fields. Also, fumigate infested fields thoroughly with effective nematicides such as EDB, DD, or 1,3–D.

Effective control has also been achieved by allowing the potatoes to dry out in the field.

Root-Knot Nematodes

Causal agent: Meloidogyne spp.

Root-knot nematodes are soil inhabiting and parasitize potato roots and tubers. The destruction caused by these pests is especially serious in the Western States; they are also prevalent in warm areas. Root-knot nematodes have been classified in the older literature under various names, especially *Heterodera marioni* (Cornu) Goodey. The new literature, however, ascribes these organisms to *Meloidogyne* spp. Two important species known to parasitize potatoes are *M. hapla* Chitwood and *M. incognita* Chitwood; however, other species may also attack potato.

The first symptoms produced by species of this nematode are small galls or knots on the roots, followed by excessive branching of these roots. The larvae feed along the surface of the roots. They finally remain at one point, and thrust repeatedly at this point with a stylet until the cell wall breaks down and entrance is gained to the cortex. The point of rupture attracts other larvae, a mass of which enters the root at the rupture spot. After entering the root, the larvae move through the cortex, intercellularly or intracellularly, until they reach the pericycle. Here an individual larva begins to feed, piercing the cell walls with its stylet. It will feed on five or six cells adjacent to its head. These cells become giant cells; where cell walls are broken down, these cells are surrounded by a giant cell wall. The giant cells in the stele cause disruption of the xylem and irregular xylem elements develop. If the attack is light, roots, although galled, can continue to grow and put out extensive branches.

The aboveground symptoms of infected plants are related to the root damage. The plants are stunted, the leaves sometimes are discolored, there is some wilting, and, in severe cases, premature death of the plants occurs. Yields are reduced. Soil temperature and soil moisture affect the extent of invasion and the severity of root and tuber galling.

Tubers that are heavily infested develop a

rough, wartlike surface with brown specks under the roughened surface. Lightly infested tubers are usually symptomless.

Control

The most effective control measure for root-knot nematodes is to fumigate infested soil with a good nematicide, such as EDB, DD, or DD-Mencs.

Other control measures are to (1) avoid use of nematode-infested land when planting, (2) use nematode-free seed, (3) practice crop rotation, (4) employ good sanitation, and (5) summer-fallow the land to kill the weed hosts.

INSECT INJURIES

There are more than 100 known pests of the potato in the United States. Recommended methods for control of these insects on potatoes are given in U.S. Department of Agriculture Farmers' Bulletin 2168. Descriptions of the insects, together with their life histories and habits, can be found in U.S. Department of Agriculture Handbook No. 264.

In this Handbook, we treat only the problems caused by the feeding of the potato leafhopper and the potato psyllid. In table 1 you will find reference to the injuries discussed here, the particular insect responsible for each injury, and the recommended control.

Hopperburn

Cause: Feeding of the potato leafhopper (*Empoasca fabae* (Harris)).

The potato leafhopper has been implicated as the usual cause of the type of tipburning known as hopperburn. The effect of the feeding of the insect on potato leaves causes this leaf injury.

The symptoms of hopperburn are similar to those of tipburn described on p. 66. A gradual dying and a blackening of the tip and margins of the leaflets occur. The affected margins roll or curl upward, the leaves dry up, and all dead tissue becomes very brittle.

This insect, in feeding on the leaves, sucks the plant juice from the midrib or other veins. The browning of the leaves results from a toxic substance injected into the leaf by the feeding.

The severity of the symptoms of hopperburn is increased by high temperature and lack of moisture.

Control

The best control for hopperburn is the effective control of the potato leafhopper with a preplant soil application of a systemic insecticide, such as disulfoton. Follow this with mid- and late-season applications of a locally recommended foliar insecticide. Follow cultural practices that ensure an adequate supply of soil moisture.

Psyllid Yellows

Cause: Damage resulting from feeding of nymph of the potato or tomato psyllid (*Paratrioza cockerelli* (Sulc.)).

Psyllid yellows is a disease that occurs in Western States, often most serious in Colorado, Wyoming, western Nebraska, New Mexico, and Utah. It is caused by a toxic substance introduced by nymphs of the potato psyllid during its feeding. The damage caused by this substance results in a complete change in the metabolism of the plant.

The symptoms on damaged plants resemble those that might be caused by a virus or *Rhizoctonia* or both. The disease is systemic and the physiology of the entire plant is affected. The first symptoms are yellowing of the margins and an upward rolling of the basal parts of the smaller leaflets on young leaves of affected plants. The rolled leaves usually turn reddish yellow; leaves of some varieties turn purple. Older leaves of a diseased plant roll upward over their midribs. The whole plant then appears to be reddish yellow or purplish, according to the absence or presence of anthocyanin in the leaves. Brown necrotic areas develop and cause the early death of the entire leaf. Buds above and below the ground are stimulated into growth. Above the ground, the axillary buds may develop into stalky shoots and often branch. When these shoots are fully developed, they give the plant a com-

pact, pyramidal shape. Sometimes, the axillary buds develop into serial tubers along the entire stem. They often produce short, leafy shoots with rosettes of small leaves which, in the advanced stages of the disease, may become bright yellow.

Psyllid yellows causes a potato plant to produce an abnormally large number of underground tubers, as many as 100 on a seriously affected plant. Several tubers may develop along a single stolon; thus, many small tubers are produced. Affected plants on which psyllids feed before tubers are set almost always produce a large number of tubers too small to be marketed. Many of these small tubers sprout and produce new plants before the mother plant dies. Unlike virus diseases, psyllid yellows is not tuber perpetuated. Although plants grown from diseased tubers are smaller and less vigorous than normal plants, they do not show the leaf rolling or other symptoms of psyllid yellows.

Control

Satisfactory control of psyllid yellows is achieved by control of the potato psyllid. Modern insecticides make this control relatively easy. Disulfoton, a systemic insecticide, is an effective control for the potato psyllid.

Some varieties, such as Irish Cobbler and Bliss Triumph, are somewhat more tolerant of the disease than other varieties.

ABIOTIC DISEASES

Plants often suffer from many disorders that are not directly attributable to parasitic agents, such as the fungi, viruses, bacteria, and nematodes or to insect feeding injuries. These disorders are abiotic, or noninfectious. They cause marked and sometimes deleterious changes in the external and internal makeup of the plant. The symptoms of these disorders are such as to justify the name of "disease."

In the potato, abiotic diseases affect skin color and tuber shape and can cause internal and external chloroses. Often, a change occurs in the gross morphology of the plant. Examples of these defects are (1) greening, resulting from excessive exposure to light; (2) second growth, caused by unevenness in the water supply during the growing season; (3) freezing injury, which can cause three distinct types of tuber injury; and (4) internal black spot, caused by bruising.

Sometimes plants suffer from nutritional deficiencies or from toxic imbalances from mineral excess. An example is zinc deficiency, which affects potatoes grown on alkali muck soils. This deficiency causes a chlorotic mottle with necrotic spots on the leaves and a dwarfing of the plant. In deficiency problems, acute symptoms are sometimes present—symptoms acute enough to remove the plant from the "normal" category. Often, however, acute symptoms are lacking, and a plant can then be considered normal.

The significant factor in all of the abiotic diseases discussed in this section of the Handbook is that they are noninfectious. The common abiotic diseases of potato and controls for these diseases are given in table 1.

Air Pollution Damage

Causes (for potato plants): The atmospheric impurities of (a) ozone and (b) PAN (peroxyacetyl nitrate).

Air pollution has become an increasingly severe problem in man's environment. The pollution of the air causes appreciable injury to many crops, including potatoes.

The atmospheric impurities that damage potatoes are ozone and PAN. Excess ozone causes a dark-brown stippling of the upper leaf surfaces of susceptible potato plants. It is most severe on the older, mature leaves and, except for yellowing of the leaves, may sometimes resemble ordinary leaf senescence (fig. 32). This damage, combined with the natural senescence of the leaves, causes the leaves to become very susceptible to leaf-spotting organisms, such as species of *Stemphylium, Chaetomium,* and *Alternaria.*

PAN produces a silvering or glazing on the

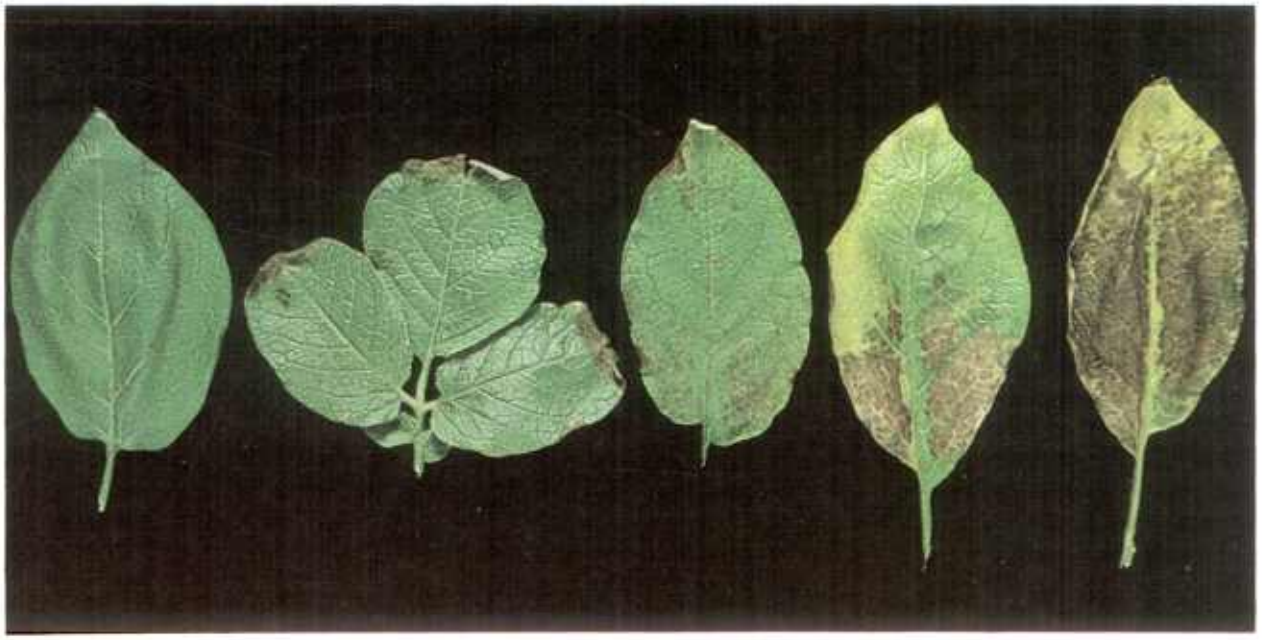

Figure 32.—Effect of ozone injury on potato leaves.

lower surfaces of affected leaves. The young, fully expanded leaves show the most severe symptoms. In extreme cases, some injury may also be visible on the upper leaf surfaces.

Control

No control for air-pollution damage is known. To avoid damage, potatoes should ideally be grown in areas that are not polluted with ozone and PAN. Varieties that resist this type of injury are being developed.

Blackheart

Cause(s): High-temperature storage; limitation of oxygen supply at high-temperature storage; combination of high temperatures and lack of ventilation.

Blackheart is a tuber condition that results from asphyxiation of the tissues of the tuber. This condition occurs in storage or transit when tubers are subjected to very high temperatures or are stored where the ventilation is so poor that the supply of oxygen is inadequate, or results from a combination of both factors.

The external symptoms of blackheart, which rarely occur, are moist areas on the tuber surface. These areas may be purplish for a short time but, characteristically, these discolored areas are brown or black. The internal symptom is a dark discoloration, grayish to purplish or inky black (fig. 33). Generally, this discoloration is restricted to the heart of the tuber. Occasionally, however, it radiates to the skin. It sometimes appears in zones in the outer parts of the tuber and is absent or less conspicuous at the center. Usually, the discolored tissues are sharply set off from healthy tissues. They are firm and leathery if they have dried out somewhat. In advanced stages affected tissues dry out and shrink, forming cavities.

When tubers are cut soon after injury, the exposed tissues are of normal color. Shortly after exposure to air, however, they turn pink, then gray, purplish, or brown, and finally become jet black. Sometimes only gray or brown discoloration is found. This occurs when tubers have been heated to above 55° C or when they have been deprived of all oxygen for considerable periods of time, as in waterlogged soils or in flooded storage pits.

Asphyxiated tissues are easily invaded by bacteria and fungi. These cause watery or slimy decay that masks the typical blackheart symptoms.

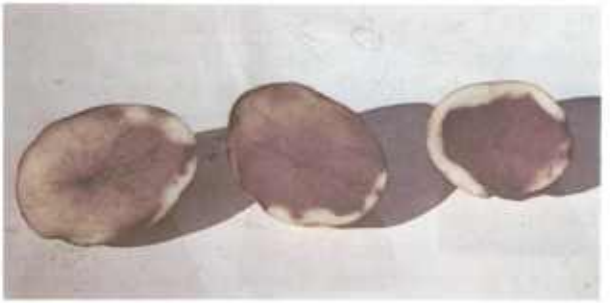

Figure 33.—Blackheart in potato tuber.

Low-temperature storage of tubers in sealed containers, where the oxygen supply is limited, will also produce blackheart.

Control

For controlling blackheart, two basic recommendations are to (1) avoid high-temperature storage and (2) provide good ventilation of storage areas. When potatoes are in transit, do not allow temperatures in heated cars to go above 16° to 21° C. To prevent oxygen shortages, do not store tubers in solid piles higher than 6 feet, even at low temperatures. During harvesting, remove tubers promptly from hot, light soils after the vines die. Also, remove tubers dug during hot weather promptly from the soil surface. Russet Burbank is a variety that resists blackheart.

Enlarged Lenticels

Cause(s): Leaving potatoes in wet soil after digging; storing freshly dug tubers in a very moist atmosphere.

The lenticels, or natural pores, of the potato tubers are ordinarily inconspicuous slits on the tuber surface. These lenticels become enlarged when potato tubers are allowed to remain in wet soil after digging, or when freshly dug tubers are stored in a place where the air is very moist.

When lenticels are enlarged, numerous scab-like openings appear on the tuber skin. The tissues around many of these small openings become raised and appear to be pushed out from below. Frequently this tissue becomes corky in appearance. The damage done to the tuber by enlargement of the lenticels is solely to its appearance.

Control

For controlling enlarged lenticels, remove tubers promptly from the field and store them under proper humidity-controlled conditions. In the West, do not overirrigate the fields. Level the land to eliminate some spots that might be wetter than the rest of the field.

Feather and Scald

Causes: Rough handling of immature tubers; effect of environmental conditions on immature, feathered tubers.

Feather and scald are skin defects of the immature tuber.

When immature tubers are handled roughly, the epidermis, or "skin," scuffs and can be rubbed off very easily. The name "feather" or "feathering" has been applied to this defect. No vine symptoms occur with this defect because it arises at or after harvest.

The condition of "scald" is induced by the action of sun and wind on the immature, feathered tubers. The exposed tissue often turns dark and presents a scalded appearance.

Control

For controlling feather and scald, avoid harvesting immature tubers. Handle tubers carefully when they are harvested. Do not expose harvested tubers to wind or sun for extended periods during the harvest operations.

Fertilizer Burn

Cause: High-nitrogen concentrations near seed pieces and roots.

In certain potato-growing areas, particularly those where furrow irrigation is used, high concentrations of nitrogen in contact with seed pieces and potato roots can cause seed-piece decay and root burning.

Seed-piece decay causes missing hills. When potato roots suffer from fertilizer burn, there is a lack of vine growth and darkening of vine color. There is also a general lack of vigor in the period of early growth.

Control

For controlling fertilizer burn, place highly concentrated nutrients far enough from the potato seed piece to avoid nijuring the roots, but close enough for irrigation water to dissolve the nutrients and move them into the root zone. The water level must be considered in relation to fertilizer placement so that soluble nutrients are made available to the plant.

Check with soil specialists in your region for local recommendations on methods of avoiding fertilizer burn.

Freezing, Frost or Freezing Necrosis, and Low-Temperature Injury

Cause: Ice formation in tissues of tubers exposed to freezing temperatures; extended low-temperature storage.

Exposure of tubers to freezing temperatures leads to ice formation in the tuber tissues and causes a variety of symptoms usually known as freezing injury. "Freezing" is the name given to the external type of injury where the symptoms are general and externally apparent. "Frost or freezing necrosis" is localized internal injury, visible only when the tuber is cut. Both types of injury are detected only after the tuber has been thawed. "Low-temperature injury" occurs in storages where tubers are exposed to temperatures that are low but not low enough to cause ice formation for an extended period of time.

Tissues killed by freezing look wet when they thaw. They usually become infected with bacteria, which cause a foul-smelling, slimy, or sticky rot if the tissues thaw in a warm, humid atmosphere. If the tissues thaw in cold or dry air, they dry into either a mealy or a tough, leathery, granular, chalky mass. When one side of a tuber is frozen, the frozen area frequently is sharply set off from the unaffected area by a purplish or brown line of corky tissue. Rot caused by *Fusarium* spp. often sets in before the unaffected cells are cut off by the corky layer.

Frost or freezing necrosis (called freezing necrosis in the following description) is marked by several types of internal discoloration. One, the ring type, is limited to the vascular ring (fig. 34*A*) and to the tissues that immediately adjoin the vascular region. Another, the net type, is marked by a blackening of the vascular tissue and of the fine strands that extend from the vascular tissue into the interior pith and to the outer tissues (fig. 34*B*). A third type of discoloration is the "blotchy" type. Blotchy discoloration is marked by irregular patches ranging in color from opaque gray or blue to sooty black. These patches may occur anywhere in the tuber. Blotchy discoloration is the only form of freezing necrosis that may be visible externally, particularly in tubers with white skins. Tubers affected with one or all types of freezing necrosis generally shrivel or wilt more than nonaffected tubers. However, excessive shriveling is not a conclusive sign of freezing necrosis. Also, it is very difficult at times to distinguish frost necrosis from several other disorders that show internal discolorations such as net necrosis and stem-end browning. Tubers showing severe freezing necrosis should not be planted because they usually rot in the soil before sprouting.

When tubers are exposed to temperatures that are low but not low enough to cause ice formation, starch is converted to sugar at a more rapid rate than it is used in respiration. As a result, the tubers develop a sweet taste. Tubers thus affected can be reconditioned by being stored at warm temperatures (10° to 16° C) before being cooked. However, most varieties subjected to low temperatures cannot be processed into potato chips satisfactorily.

Control

For controlling frost or freezing necrosis, avoid field frost by harvesting the crop and storing it before the ground freezes. Prevent excess sugar accumulation, internal discoloration, and freezing by storing potatoes at 2° to 5° C. During processing, hold potatoes at temperatures between 7° and 21°.

Heat and Drought Necrosis

Cause: Leaving tubers in hot soils after vines begin to die.

Heat and drought necrosis occurs in tubers that are grown in light, sandy soils in the hot, arid potato-growing regions of the United

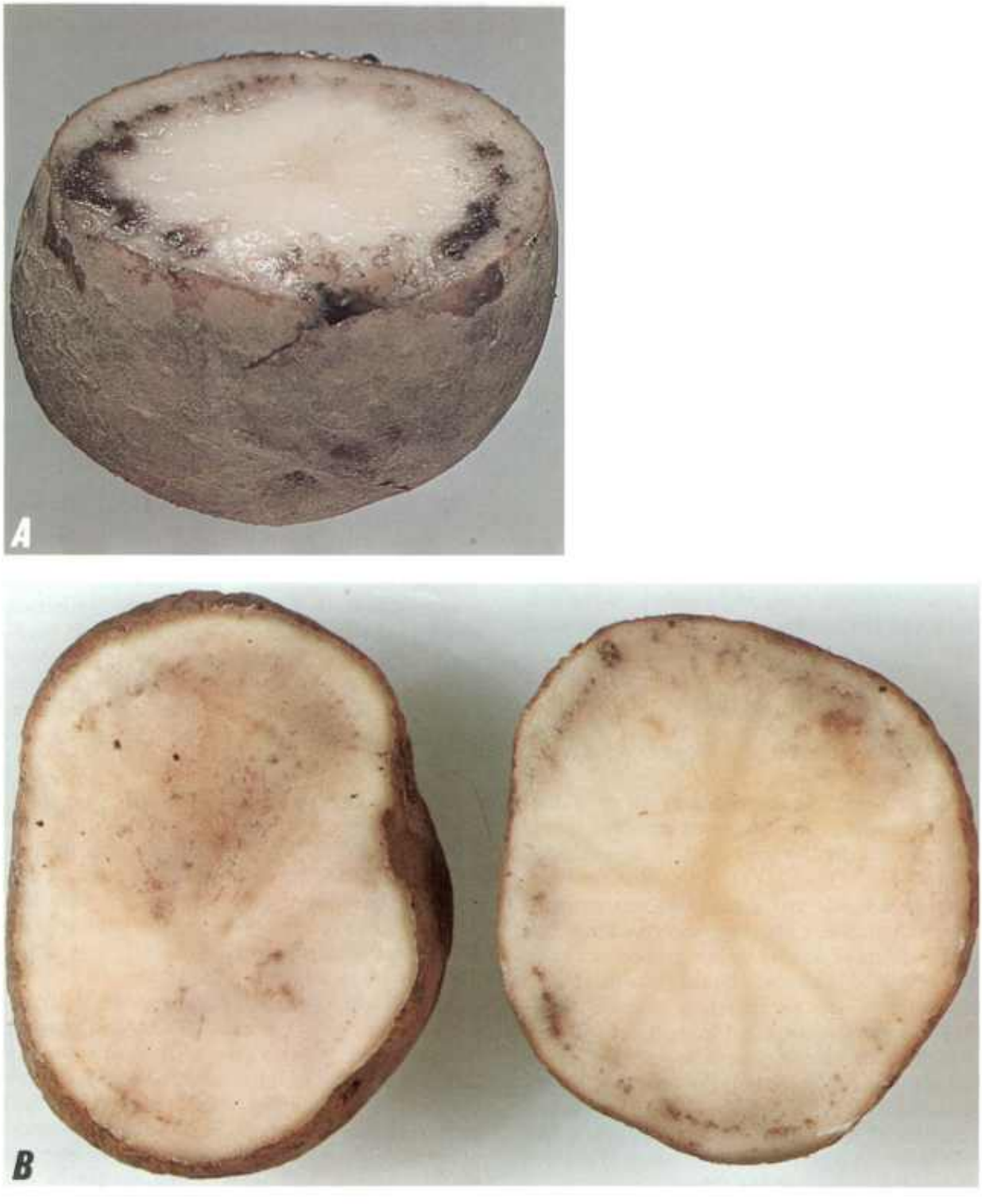

Figure 34.—Freezing necrosis injury: *A*, Vascular ring type; *B*, net type.

States. This necrosis has been found in the early crop grown in the volcanic ash soils of Idaho. It occurs in tubers that are left in the hot soils after the vines begin to die.

In tubers affected by heat and drought, a golden-yellow to brown discoloration of the water vessels occurs. This discoloration is most pronounced in the vascular ring tissues at either the stem end or the bud end, and also in the tissues between the ring and the tuber surface. At first, the discoloration is restricted to the vascular tissue. Later, it spreads slightly to surrounding tissues and the color deepens from golden yellow to a light to dark brown. Affected tissues die. When the tuber is cut, the discolorations are seen to derive from discolored strands that impart a dark hue to the tissue just above them instead of from a solid dark mass of tissue.

Control

Control heat and drought necrosis by keeping the soil moist, cool, and shaded. If the soil is light and the weather hot, dig tubers as soon as the vines begin to die.

Hollow Heart

Cause: Environmental conditions favorable for rapid growth.

"Hollow heart" is the name given to a conspicuous cavity at the center of a potato tuber. It is a defect that is prevalent in large, oversized tubers.

Usually no external symptoms are evident, and plant symptoms are completely lacking. The defect consists of a more or less irregular cavity in the center of the tuber. Usually, no discoloration occurs in the surrounding tissues. Occasionally, however, adjacent cells are brownish and corky (fig. 35). Research has shown that a relationship exists between the shape of tubers and the formation of the hollows. The stellate formation of the hollows is a compensation of tissue tensions that result from unequal growth in three dimensions of the tubers.

Hollow heart is not a decay and the condition has no effect on plants grown from affected tubers. However, affected stock is undesirable for eating.

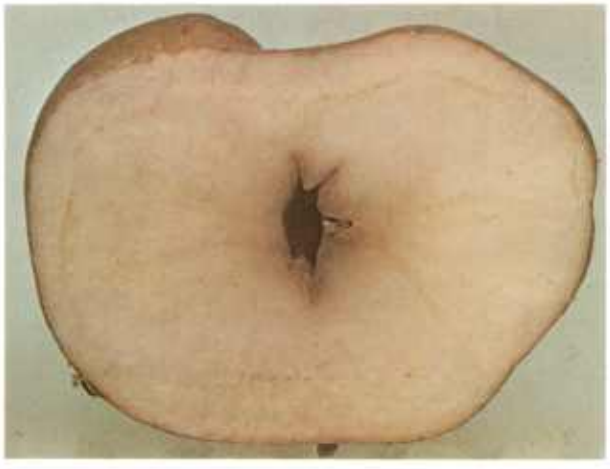

Figure 35.—Potato tuber showing hollow heart.

Control

To control hollow heart, space potatoes closely. Close spacing prevents rapid and uneven growth of the tubers and tends to prevent internal splitting. Apply adequate but not excessive amounts of fertilizer and irrigation water to the crop. Limit the formation of oversized tubers by vine killing. Avoid planting those varieties that develop this defect, such as Chippewa, Katahdin, Mohawk, Irish Cobbler, Sequoia, Russet Rural, or White Rose.

Internal Black Spot

Cause: Bruising. Predisposing factors include loss of tuber turgidity because of dry soil and high carbon dioxide/low oxygen ratios in storage atmosphere.

Internal black spot is a defect that often develops in tubers after harvest.

This defect is characterized by the development of light-gray to coal-black lesions beneath the skin (fig. 36). These lesions are usually found at the site of a bruise or a pressure mark that did not break the skin. The severity and extent of the defect is influenced by several other factors, such as low potassium levels, low starch content, and lack of cell turgor in the tuber. Other causes that contribute to this defect are high atmospheric carbon dioxide/low oxygen ratios in the storage area, high storage temperatures, lack of tuber maturity upon

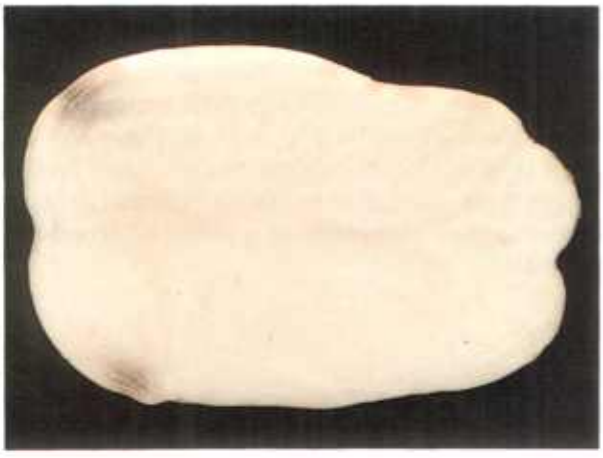

Figure 36.—Internal black spot in the tuber.

storage, and the effects of top killing of the vines.

Control

The prime measure for control of internal black spot is to handle the tubers carefully to avoid bruising them. Irrigate dry soil to maintain tuber turgor. The tubers should also be handled so as to prevent loss of moisture. Harvest the tubers when they are mature and store them at 21° C for 2 days, then store them at about 5° to minimize losses from black spot.

Plant resistant varieties. Pontiac appears to be resistant, but Teton, Ontario, and White Rose varieties are very susceptible.

Internal Brown Spot

Cause(s): Hot, dry weather during the growing season; lack of soil moisture.

Internal brown spot is a disorder of potatoes that is frequently called by other names: Internal necrosis, internal brown fleck, internal rust spot, internal browning, and, in part, sprain (not, however, spraing).

Internal brown spot is characterized by irregular, dry, brown spots or blotches scattered throughout the flesh of affected tubers (fig. 37). These spots are not restricted to the water vessels, as in vascular diseases, but are often found in the central parts of the tubers. The spots are groups of dead cells that are free from bacteria and fungi. No vine or foliage symptoms or external tuber symptoms are associated with this defect.

Internal brown spot occurs most often in tubers grown in sandy soils; however, it has also been reported in potatoes grown on muck soils. Factors that are implicated with this defect are lack of available soil moisture, particularly late in the growing season; lack of water at some critical stage in the growth of the plant because of either poor soil texture or alternating wet and dry weather; and excessive evaporation of moisture from the foliage.

Control

Favorable cultural conditions, such as proper irrigation practices and covering the tubers with 2 inches or more of soil, reduce the severity of internal brown spot. Avoid planting varieties such as Katahdin, Green Mountain, Ontario, Pawnee, and Russet Rural that are susceptible to this defect.

Lightning Injury

Cause: Lightning striking a field.

During electrical storms, lightning occasionally strikes an area in a potato field. When this phenomenon occurs, most of the plants in a more or less circular area are killed (fig. 38). The tubers are "cooked" in the soil. Organisms present in the soil then break down the tubers within a few days after they are struck.

Figure 37.—Internal brown spot injury in Irish Cobbler.

Control

None.

Figure 38.—Lightning injury to potato plants.

Second Growth (Knobby Tubers)

Cause(s): Uneven growing conditions at mid-season; possibly also giant hill (bud mutation).

Second growth, often called knobby tubers, is a condition produced in potatoes most frequently when dry weather prevails during mid-season and is followed by a rainy period. Some varieties, particularly those that produce long tubers, are more subject to knobbiness than other varieties.

The second growth occurs on the main or primary tuber at sites where eyes are located. These growths vary considerably in shape and size (fig. 39). No internal tuber symptoms or plant symptoms occur. In dry weather, tuber growth ceases. After a rainy period, growth begins again. When tuber growth resumes, it is not uniform. Proliferations, or knobs, occur at the site of one or more eyes. Sometimes affected tubers have pointed ends. This abnormal growth response may also occur as a result of irregular irrigation.

Control

To control second growth (knobby tubers), provide uniform growing conditions throughout the growing season. Proper irrigation is important in the West. Plant varieties that produce round tubers. Avoid planting those varieties that tend to be excessively knobby, such as Green Mountain and Russet Burbank.

Spindling Sprout (Hair Sprout)

Cause: Tuber weakness.

Spindling sprout, or hair sprout, is a tuber defect that is characterized by the formation of abnormally slender and feeble sprouts.

The defect is frequently nontransmissible and is then primarily an inherent weakness of the tuber. However, the presence of other specific disease organisms, such as the leafroll virus and the witches' broom virus, may or may not be associated with the defect. When the abnormality is of a nontransmissible type, some of the sprouts have a diameter about one-half to one-fourth that of normal sprouts (fig. 40). Plants developing from such sprouts bear small tubers, each of which weighs only an ounce or less. However, when these tubers are planted, they produce stocky, vigorous shoots. These shoots produce plants that bear normal tubers weighing from 5 to 7 ounces. When healthy tubers are grafted with plugs from tubers affected with spindling sprout, the healthy tubers do not develop spindling sprout.

Control

Where spindling sprout has been prevalent, presprout the seed to determine whether spindling sprout is present in the seed stocks.

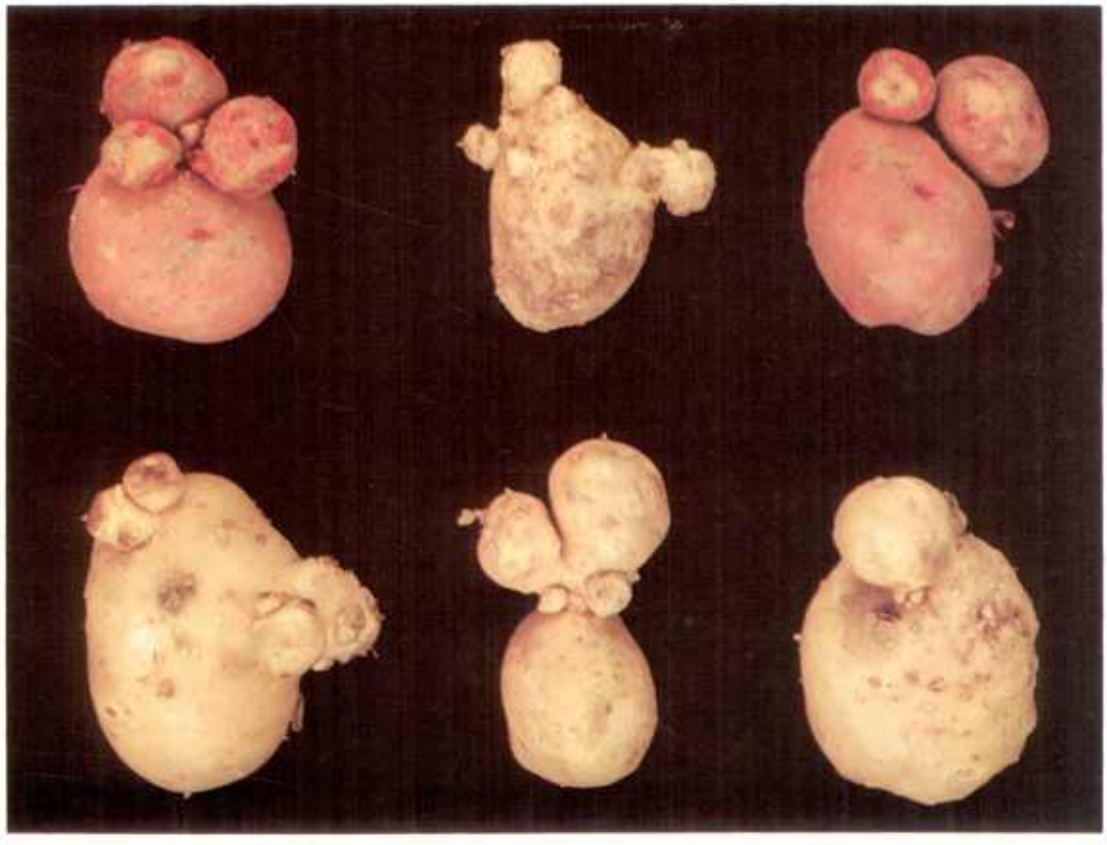

Figure 39.—Knobby tubers, or "second growth" condition.

Sprout Tubers (Secondary Tuber Formation)

Cause: Excessive concentration of cell sap in tubers.

"Sprout tubers," "secondary tuber formation," or "potatoes with no tops" are common names for second growth. This irregularity occurs when the rest period is completed in the spring after the seed is planted or when conditions are unfavorable for normal vegetative growth.

A poor field stand is the first sign of secondary tuber formation. Although seed pieces are firm, a small, marble-sized potato has grown directly from an eye. If sprouts form, they are short and they terminate in new tubers (fig. 41).

Control

To avoid formation of sprout tubers, place seed potatoes in cool storage. Avoid unusually long storage in light at temperatures of 18° to 20° C as these storage conditions predispose the potato to form secondary tubers. Practice late planting.

Stem Streak Necrosis

Cause: High levels of soluble manganese in soil.

An unusual necrotic streak of potato stems has been noted when plants are grown on acid soils and is associated with high levels of soluble manganese in the soil.

The symptom of this necrosis is the presence of irregular necrotic streaks on affected stems and petioles (fig. 42). The lower leaves turn yellow and drop. This latter symptom resembles, and can be mistaken for, current-season infection with potato virus Y.

Four types of stem streak caused by four different factors are recognized. One type is called late-blight streak. Two other types of streak, Verticillium streak and virus Y streak,

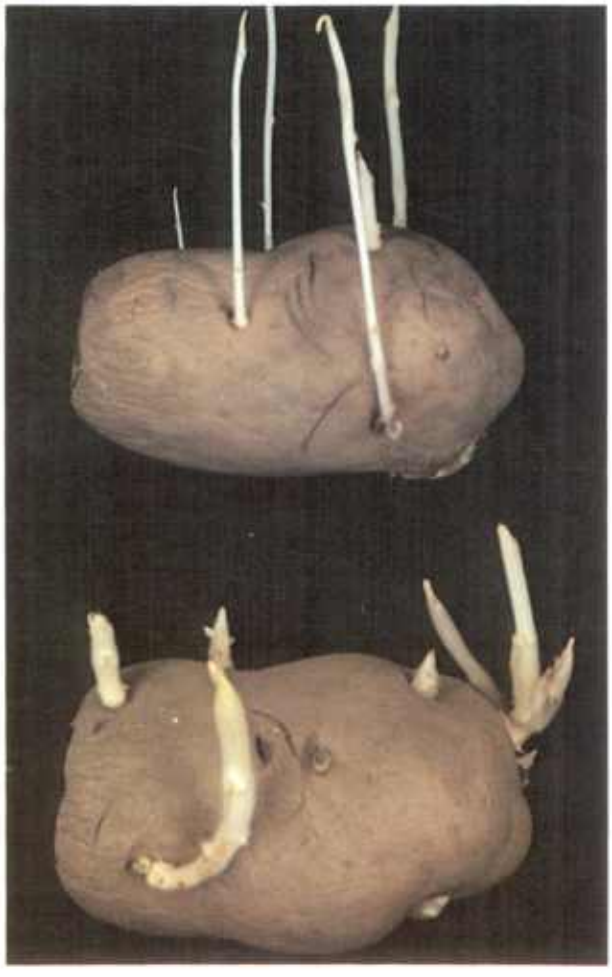

Figure 40.—Tuber showing slender feeble sprouts characteristic of spindling sprout (top); normal sprouts on healthy tuber (bottom).

are caused by pathogens. The fourth type, stem streak necrosis, is caused by manganese toxicity. When manganese levels are high in the soil, the Verticillium-streak symptoms are also more pronounced.

Control

To control stem streak necrosis (1) apply lime, (2) add fertilizers to the soil, and (3) use resistant varieties. Varieties resistant to streak are Canso, Green Mountain, and McIntyre. Cherokee, Irish Cobbler, Merrimack, and Sebago are very susceptible to streak and should be avoided when potatoes are to be grown on acid soils or where levels of manganese are high.

Sunburn (Greening)

Cause: Exposure of tubers to sunlight or artificial light.

Sunburn, or greening, results from the exposure of the tubers to sunlight during growth or after digging, and to artificial light during holding periods on display stands. This exposure can occur in the field, in transit, in storage, or in the retail store. Sunburn does not kill the affected tissues.

After exposure for 2 days or longer to either natural or artificial light, a green pigment develops in the exposed tubers and they become light green to dark green (fig. 43). The skin and the flesh, from 2 to 10 mm in depth, are affected. The outer tissues become deep green and those underlying become greenish yellow or deep yellow in color. Greening and yellowing of tubers occur more readily if the tubers have been dug before maturity. These immature

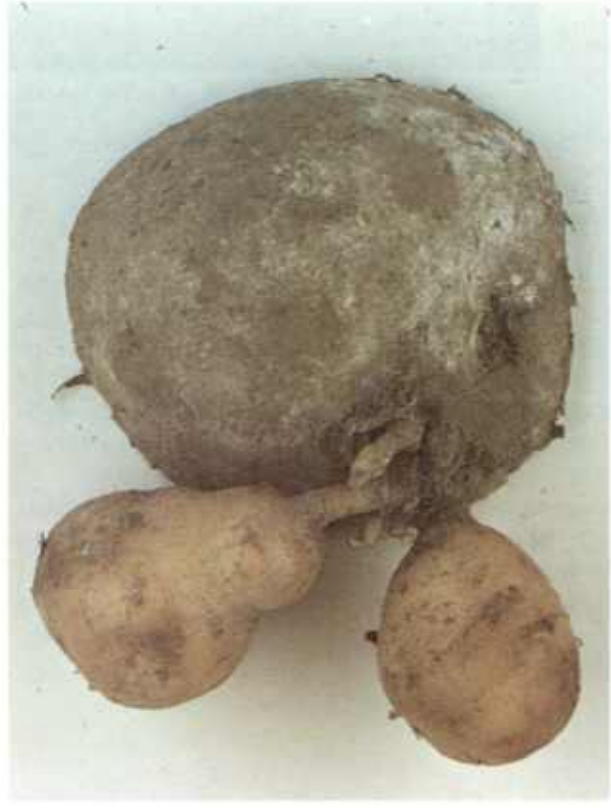

Figure 41.—Potato tubers showing secondary or sprout tubers.

Figure 42.—Necrotic streak of potato stem (right); normal (left).

tubers also have a tendency to shrivel more readily than mature tubers with well-developed cork layers.

Chlorophyll and solanine are produced in the flesh of tubers exposed to light for extended periods of time. The green tubers acquire a bitter, pungent taste. If eaten in quantity, they may be poisonous. Solanine is the bitter and poisonous component; chlorophyll is tasteless and harmless.

The green pigment develops faster and also fades faster at 21° than at 2° C. Waxing the tubers will mask greening but will not prevent it. Potatoes that are washed will turn green more rapidly than unwashed potatoes.

Control

To control sunburn in the field (1) practice proper hilling and (2) control Rhizoctonia infection. To prevent greening (1) store potatoes in complete darkness, even under home conditions, and (2) market the tubers in light-proof containers and rotate potatoes on display in the market.

Potato breeders are now working to develop new varieties that will resist greening.

Sunscald

Cause: Exposure of plant to hot sun and drying winds following cloudy weather.

Sunscald is an environmental effect that causes parts of young, tender leaflets to become light green, wilt, then dry up and become tan. This condition is sometimes mistaken for late blight infection, but no parasitic organism is involved. Sunscald also affects tubers that are exposed to the sun during growth or after digging, either in the field, in transit, or in storage.

In tubers, frequent exposure to sunlight and the resulting high temperatures result in the killing of some tuber cells. Often affected tubers become watery and turn brown to a considerable depth or throughout the entire tuber. Some freshly scalded areas have a blisterlike appearance externally and a metallic color. The underlying tissues are watery. Such areas may dry out and become either chalky and granular or hard and leathery. Generally, affected areas are attacked by bacteria that cause foul-smelling rots.

Control

Prevent exposing tubers to the sun for prolonged periods of time.

Tipburn

Cause: Excessive loss of moisture during hot, dry weather.

Tipburn is a condition that develops when excessive amounts of moisture are lost from plant tissues during periods of hot, dry weather.

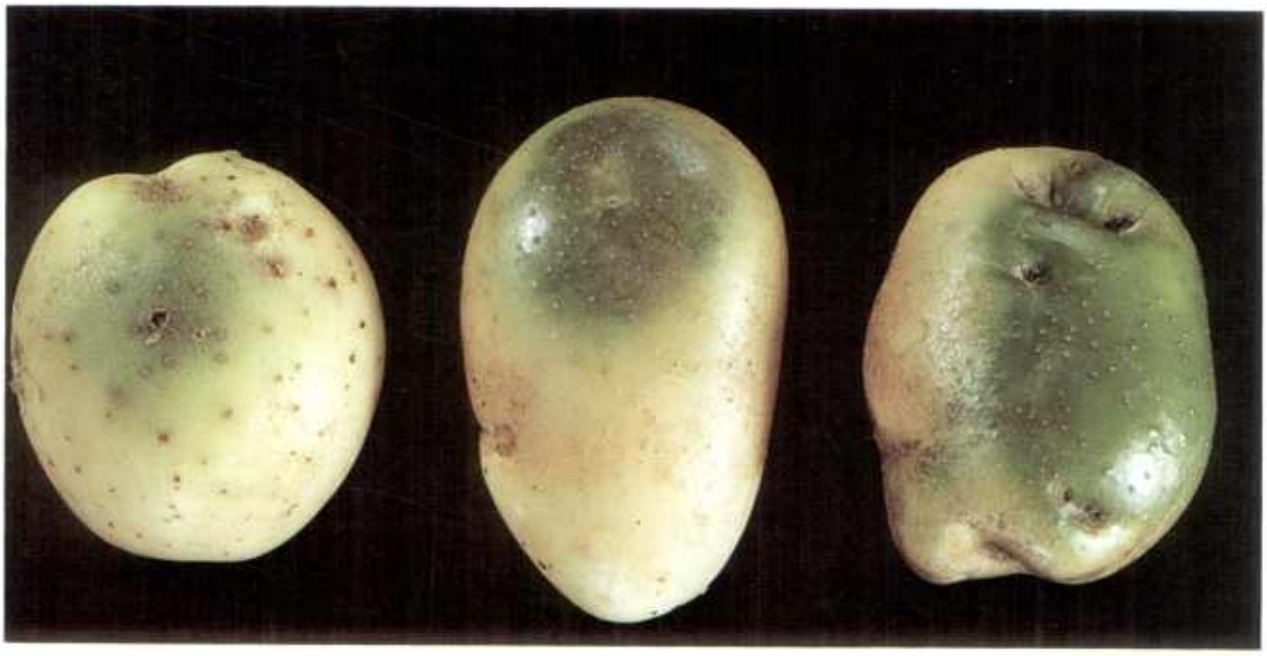

Figure 43.—Greening of potato tubers after prolonged exposure to light.

The symptoms of tipburn are a yellowing of the tips and margins of the leaflets, then a gradual dying and browning or blackening of these parts (fig. 44). More than half of the leaf surface may die. These affected margins roll upward, and all the dead tissue becomes very brittle. Affected leaf tissue is often torn. Plants that are weakened by the presence of disease entities, such as the leafroll virus and the spindle tuber viroid, are more susceptible to tipburning than healthy plants.

The withdrawal of water from plant tissues during hot, dry weather may eventually cause the death of leaves affected with tipburn unless the desiccated leaf condition is relieved by rainfall or by a reduction in temperature.

Control

Any measure that conserves soil moisture is helpful in reducing tipburn. Effective insect control is also recommended, particularly for the control of the potato leafhopper (*Empoasca fabae*). (See discussion on Hopperburn.)

Xylem Ring Discoloration

Cause: Rapid killing of the potato vines.

In other sections of this Handbook, various types of stem-end discoloration of potato tubers are described and discussed (for example, phloem (net) necrosis, stem-end browning, freezing or frost necrosis, and Verticillium wilt).

Another type of discoloration also occurs. This is the brown discoloration of the xylem ring that is visible in affected tubers at harvesttime. The discoloration usually extends throughout the length of the tuber. It occurs when there is rapid killing of potato vines, either by chemicals or by frost. Severe xylem ring discoloration makes potatoes undesirable for table use. Seed potatoes are not affected.

Control

To prevent xylem discoloration, avoid using chemicals that kill the vines rapidly. Use of mechanical beaters instead of chemical vine killers is increasing in popularity.

Figure 44.—Symptoms of tipburn on potato.

GLOSSARY

This glossary lists many of the terms found in potato and other disease literature; the terms are followed by short definitions or explanations. We have incorporated most of the terms used in this Handbook and have added others that have general significance or are terms related to the diseases, genera and species, spore structure, life processes, and so forth. These definitions are included as an aid to those readers with a general interest in plant pathology or a specific interest in potato diseases.[3]

abiotic—without an infectious, transmissible, or parasitic cause (see text, p. 61).

Actinomycetales, bacteria—an order in which the Streptomycetaceae is one family. In the Streptomycetaceae, the organisms [=*Streptomyces*] have mycelium, conidia, no arthrospores. In potato, usually associated with the fungi.

Alternaria, fungus—a genus in the Moniliales; cause of early blight of potato; occurs principally as a leaf spot.

anthocyanin—a soluble, reddish-blue pigment in flowers and plants.

antibiotic—any of certain chemical substances produced by various groups of micro-organisms. These substances have the capacity, in dilute solutions, to inhibit the growth of or to destroy bacteria and other micro-organisms.

appendage—any subordinate or external organ or part.

apical—at the end of (or apex).

appressed, adpressed—closely flattened down.

arthrospore—a spore that results from the breaking up of a hypha into separate cells.

ascocarp—a sporocarp, or ascus-producing structure, in the Ascomycetes.

ascus—a saclike cell of the perfect state of an Ascomycete in which the ascospores (generally 8 in number) are produced by free cell formation.

atom—a minute particle of matter; the smallest particle in which an element can be divided without losing its identity as an element.

attenuated—narrowed; can also indicate lowered pathogenicity or virulence.

atypical—not normal.

bacterium (pl. -a)—microscopic, 1-celled organisms, multiplying by binary fission; rod shaped; fixed or free filamentous forms; motile by one or more flagella, or nonmotile. Polar flagella (*Pseudomonas*); peritrichous flagella (*Erwinia*).

bactericide—substance used to kill bacteria.

Basidiomycetes—a class of fungi in which the basidium is the structure on which the "sex" spores undergo development.

basidiospore—a spore acrogenously adjoined upon a basidium, typically following karyogamy and meiosis.

basidium—the organ or cell, usually terminal, in the Basidiomycetes, which after karyogamy and meiosis, bears the basidiospore.

biologic form, a race—biotype or a physiologic race of an organism.

biotype, physiologic race—individuals with similar genetic makeup.

Black dot—a weakly parasitic disease of potato caused by *Colletotrichum atramentarium* (Berk. & Br.) Taub.

Blackleg (of potato)—a disease caused by *Erwinia phytophthora* (Appel) Holland.

Black scurf (of potato)—a disease caused by *Rhizoctonia solani* Kühn.

blight—name given to a number of plant diseases; early blight of potato, *Alternaria solani* (Ell. & G. Mart.) Sor., and late blight, *Phytophthora infestans* (Mont.) D By.

chlamydospore—an intercalary or terminal asexual spore.

chlorophyll—the green coloring matter of plants which in the presence of sunlight converts carbon dioxide and water into carbohydrates.

class—one of the main subdivisions of a phylum.

cleistothecium—a fungus fruiting body that has no special opening; said of the ascocarp of the Erysiphaceae.

coalesce—to unite or merge into a single mass or group.

coenocytic—multinucleate or without cross walls.

Colletotrichum—a genus in the Melanconiales. A cosmopolitan *fungus; C. atramentarium* is the causal agent of a minor disease of potato called black dot.

conidiophore—a simple or branched hypha on which conidia are produced.

conidium, conidiospore—any asexual spore except a sporangiospore or intercalary chlamydospore.

cortex—a thick outer covering.

Corticium—*Rhizoctonia*.

Corynebacterium—a cosmopolitan genus of the Eubacteriales; *C. sepedonicum*, cause of ring rot of potato.

cryptogam—a plant that bears no flowers or seeds but propagates by means of special cells called spores.

cyst—a saclike structure.

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[3] For information on genera and species of the fungi, please consult:

Ainsworth, G. C. 1971. Ainsworth & Bisby's Dictionary incl. Lichens by P. W. James and D. L. Hawksworth. 6th ed., Commonwealth Mycological Institute, Kew, Surrey, England. 631 pp.

Snell, W. H., and Esther A. Dick. 1971. A Glossary of Mycology. Rev. ed., Harvard University Press, Cambridge, Mass. 181 pp.

dalton—a unit of mass convenient for the expression of the masses of atoms, being one-sixteenth of the mass of an oxygen atom. It equals approximately 1.65×10^{-24} grams.

damping-off—a rotting of plant tissue at the soil level.

differential hosts—species or varieties of plants used for determining biotypes or physiologic races of an organism.

diffuse—not concentrated; spread out.

disinfect—to inactivate or remove a pathogen from the host.

disinfest, disinfestation—to decontaminate an area; act of decontaminating.

dormant—not vegetating, inactive.

endemic—occurring naturally in a country or in a special area.

endogenous—developing from within; originating internally.

enphytotic—damage from a plant disease that occurs in constant form from year to year.

epibiotic—anything that lives on the surface of another organism.

epidemic—general and severe infection or infestation; said of a disease that is rampant.

epiphytotic—an epidemic among plants.

Erwinia—a genus of bacterial plant pathogens; *E carotovora* causes soft rot in potato and in other crops

Erysiphe—a member of the Erysiphaceae; powdery mildew.

fertile hyphae—see conidiophore.

filamentous—threadlike.

fission—complete division of an organism so that two organisms are formed.

flagellum (pl. -a)—a whiplike appendage of a motile cell.

fungicide—a chemical or other substance that causes destruction of fungi.

fungistasis—failure of spores to germinate in natural soil under conditions that one would expect would permit germination, that is, normal pH and soil temperature conditions, and so forth.

fungus—see text, p. 20.

Fusarium—a genus of fungi that has fusoid, curved, septate macroconidia; members of this genus cause wilt, dry rots, and seed-piece decay in potato.

gall—a swelling or outgrowth produced by a plant as the result of an attack by a fungus or other organism.

genome—minimum group or set of chromosomes derived from a gamete or zygote; a number of qualitatively different chromosomes that together form a unit.

genotype—the genetic or factorial constitution of an individual; group of individuals possessing the same genetic constitution.

genus—a classification of plants or animals with common distinguishing characteristics; the main subdivision of a family.

germ tube—a germination hypha.

Gram, positive or negative—said of bacteria in that they take, or do not take, Gram's stain.

granular—containing or consisting of grains or granules; covered with small particles.

greening—development of green pigmentation on the surface of tubers due to the formation of chlorophyll when tubers are exposed to light.

host—a plant or living organism that harbors a parasite.

hypha (pl. -hyphae)—one of the threads of a mycelium.

immune—exempt from infection; possessing qualities that do not allow the development of disease.

imperfect state—see state.

infection—act of entering and establishing a pathogenic relationship with the host.

infest, infestation—to overrun or contaminate an area; act of invading or contaminating.

inoculate—to inject a disease agent, a virus or the like into a plant; to put or implant bacteria or the like into the soil.

inoculum—the material used to make an inoculation, such as spores, bacteria, viruses, and so forth; sometimes called inoculant.

karyogamy—conjugation of nuclei in the process of syngamy.

larva—an insect in the earliest stage of development.

lenticel—a respiratory pore in stem epidermis of higher plants; in potato, noticeable on the tuber.

macroscopic—visible without a lens.

microscopic—visible with a lens only.

micro-organism—any microscopic or ultramicroscopic organism, that is, bacteria, fungi, virus, and so forth.

mildew—a plant disease in which the pathogen is seen as a growth on the surface of the host; powdery mildew of potato is caused by a member of the Erysiphaceae.

mold—a growth on the surface of organic matter, caused by fungi, particularly when dampness or decay is present.

motile—capable of or exhibiting spontaneous motion.

mottle—a blotch, streak, or spot.

mycelium (pl. -a)—the collective term for a group or mass of hyphae or fungous filaments, the thallus of a fungus.

mycoplasma (pl. -a)—a term that properly signifies members of the genus *Mycoplasma;* sometimes applied to all members of the class Mollicutes, which contains one order (Mycoplasmatales), two families (Mycoplasmataceae and Acholeplasmataceae), and two genera (*Mycoplasma* and *Acholeplasma*) at present. Mollicutes are minute, highly pleomorphic, plastic organisms that are bounded by a single, triple-layered "unit membrane," can be cultivated in cell-free media, and have no history of reversion to or derivation from a bacterial parent. These entities act as etiologic agents of viruslike diseases, that is, potato witches' broom. Remission of mycoplasma-caused diseases is achieved with tetracycline antibiotics.

mycotic—caused by fungi.

necrosis—death of plant cells, especially when the tissues become dark in color; a symptom of fungus infection.

nematode (plant)—a stylet-bearing member of the Phylum Nematoda (Roundworms) parasitizing plants and commonly microscopic in size.

net necrosis—first-year symptom of tuber infection with leafroll virus.

nucleic acid—any of several acids found in cell nuclei.

obligate parasite—an organism that has not been cultured on laboratory media; living only as a parasite.

oogonium—the female sex structure of the Oomycetes.

Oomycetes—Phycomycetes that have oospores.

oosphere—a female gamete; the "egg" of the oogonium.

oospore—the resting spore from a fertilized oosphere.

order—a subdivision within a class, itself divided into families.

parasite—an organism living on or in its host, from which also it gets its food; a living organism.

pathogen—a parasite or causal agent able to cause disease in a particular host or range of hosts.

pathogenicity—the condition of being pathogenic.

perfect state—see state.

pericycle—the outer layer of the stele in the root and stem of most plants.

periderm—the outer layers of cortical tissue; the epiphloem.

peritrichous—having hairs, or flagella, over the entire surface.

petiole—the stem of a plant leaf.

phloem—the hollow tissue which dissolved foods flow from one part of a vascular plant to another.

phloem necrosis—see net necrosis.

Phoma—a genus of fungi in the Sphaeropsidales. It is a cosmopolitan fungus with some species pathogenic on potato; some species have different ecological patterns.

Phycomycetes—a heterogenous group of fungi in which the mycelium (if present) is coenocytic, or has only a few septa.

phylum (pl. -a)—a main division of the plant or animal kingdom.

physiologic race—see biotype.

Phytophthora— a genus of fungi in the Peronosporales. *P. infestans* is an important pathogen of potato, cause of late blight of potato.

phytotoxic—toxic to plants.

pith—soft, spongy tissue in the center of certain plant stems.

polar—at the ends or poles.

pore—a minute opening such as the lenticels and stomates in plants.

Pseudomonas—a genus of bacteria in the Pseudomonadaceae. Cosmopolitan organisms. *P. solanacearum* causes bacterial wilt and brown rot of potatoes.

pustule—a blisterlike spot or spore mass, frequently erumpent.

pycnidium (pl. -a)—the fruit body of the Sphaeropsidales, frequently globose or flasklike.

Pythium—a genus of fungi in the Peronosporales; causes leak disease of potato.

race—see biotype.

resistance—ability of the plant to ward off disease; insusceptibility.

Rhizoctonia—*R. solani* causes black scurf of potato.

rugose—wrinkled.

saprophyte—an organism that uses dead organic material as food.

scab—a disease characterized by scablike lesions; *Streptomyces scabies* (Thaxt.) Waks. & Henrici causes common scab of potato; *Spongospora subterranea* (Wallr.) Lagerh. causes powdery scab of potato.

scabrous—rough.

Sclerotinia—a genus of fungi in the Helotiales. These fungi are cosmopolitan and plurivorous; *S. sclerotiorium* (Lib.) D By. (=*Whetzelinia sclerotiorum* (Lib.) Korf & Dumont) causes stalk break of potato.

Sclerotium—a genus of fungi in Mycelia Sterilia. *S. rolfsii* causes Sclerotium rot (southern blight) of potato.

sclerotium (pl. -a)—a firm mass of hyphae that normally has no spores in or on it. It may produce a fruit body.

screening—a method for testing the reaction of a host to invasion of an organism; used also for tests made to judge the particular property of a chemical substance.

sensitive—keenly susceptible to stimuli; a marked reaction to attack of a given organism.

septum (pl. -a)—a cell wall, a division in the hyphal chain.

simple—unbranched.

soft rot—a decomposition of the host caused by fungi or bacteria; *Erwinia carotovora* (L. R. Jones) Holland causes soft rot of many vegetables, including the potato.

soil fungus (pl. -i)—organism present in the soil, capable of rotting organic residues; some may be pathogenic. Soil fungi involved in complex biological relationships between various organisms in the soil and between these organisms and higher plants.

solanine—a poisonous alkaloid found in potato plants.

species—a division of a genus; one type of plant, animal, or microscopic organism.

Sphaeropsidales—an order comprised of several families in which the pycnidia are usually globose, coriaceous to carbonaceous as in the Sphaeropsidaceae of this order (Fungi Imperfecti).

sporadic—happening from time to time; isolated in occurrence, or in isolated instances.

sporangiophore—a sporophore that supports a sporangium.

sporangiospore—a spore produced in a sporangium.

sporangium—an organ that produces endogenous asexual spores.

spore—a reproductive structure in the cryptogams.

sporophore—a spore-producing structure; a structure that bears spores; term applied to a conidiophore.

sporulate—to form spores.
sprain—an internal condition of tubers characterized by the presence of rusty brown pockets of dead cells in the flesh of the tuber.
spraing—A Scottish dialect word meaning "streak"; describes the form in which the brown, or almost black, necrotic tissue is arranged in thin curved lines that do not follow the vascular ring; lines are sometimes wavy, sometimes form parts of concentric circles. Also refers to the necrotic patches in the flesh of the tuber caused by soilborne viruses (tobacco rattle virus or potato mop top virus).
state—a phase in the life cycle of an organism; perfect state is the state in which sexual spores are formed; imperfect state is state in which asexual spores or no spores are produced.
sterile—free from living micro-organisms; unable or failing to bear spores or a fruit body.
stippled—the effect in nature of being marked with small dots or points.
stolon—a trailing branch or shoot which, in the potato, is called an underground stem and which subtends the tuber from its end.
stoma (pl., stomata)—a breathing pore in the epidermis of plants through which there takes place an exchange of oxygen and carbon dioxide between air spaces inside the leaf and air outside the leaf.
Streptomyces—a genus in the Actinomycetales; forms conidia in chains. *S. scabies* causes scab in potato. Some species in this group yield antibiotics.
striate—grooved or furrowed.
stroma (pl., stromata)—a mass of vegetative hyphae.
stylet—any pointed bristlelike protrusion; mouth part of parasitic nematode.
suberin—a waxy or fatty substance contained in cork.
suberize—to change into cork by the formation of suberin in the cell walls.
susceptibility—lacking immunity to a disease or a pathogen.
swarmspore—see zoospore.
syndrome—a number of symptoms occurring together and characterizing a specific disease.
syngamy—sexual reproduction; fusion of gametes.
synonym—another name for a species, either for a plant or a pathogen.
tolerant—giving little reaction to infection by a pathogen.
tuber—a short, fleshy underground stem bearing minute scale leaves each having a bud in its axil potentially able to produce a new plant.
tuber-perpetuated—said of a plant that can be cultivated asexually by a plant part, namely, a tuber; also any disease entity carried through the tuber to succeeding generations.
unipolar—at one end only, said especially in reference to a bacterial cell.
viroid—a term now used to denote a novel class of subviral agents which are characterized by the absence of a dormant phase (virions) and by genomes that are much smaller than those of known viruses. They introduce only a small amount of genetic information into their hosts yet are able to replicate and to incite diseases in certain organisms.
virulence—the degree or measure of pathogenicity.
virulent—strongly pathogenic.
virus—see text, p. 44.
Whetzelinia sclerotiorum—as cause of stalk break of potato, see *Sclerotinia*, ibid.
wilt—disease condition in plants caused by certain fungi or bacteria that is characterized by wilting of the leaves.
xylem—the woody tissue of a plant; part of the vascular bundle that gives firmness and conducts moisture.
zoosporangium—a sporangium that produces zoospores.
zoospore—a motile sporangiospore that has flagella; a swarmspore.

HELPFUL MEASUREMENTS

LIQUID MEASURE (American to metric):

		Fluid ounce	*Milliliter*	*Liter*
80 drops	= 1 teaspoon (tsp)	--	=5	--
3 teaspoons	= 1 tablespoon (tbsp)	=½	=15	--
2 tablespoons	= 6 teaspoons	=1	=30	--
16 tablespoons	= 1 cup	=8	=236	=0.236
1 pint	= 2 cups	=16	=473	=0.473
1 quart	= 2 pints	=32	=946	=0.946
1 gallon	= 4 quarts	--	=3,785	=3.785

SOLID MEASURE: (American to metric)

1 dram	= 27.34 grains		=1.772 grams
16 drams	=437.5 grains	=0.06 pound = 1 ounce	=28.35 grams
16 ounces	=7,000 grains	=1 pound	=453.59 grams
100 pounds		=1 hundredweight	=45.36 kilograms

(grain = 0.0648 gram)

SQUARE MEASURE:

160 square rods = 4,840 square yards, or 43,560 square feet, or 1 acre, or 0.4047 hectares.

640 acres = 1 square mile, or 259 hectares, or 2.59 square kilometers.

LAND MEASURE:

1,549.9 square inches	=	1 square meter	= 1 centiare
119.6 square yards	=	1 are	= 100 centiares
2.471 acres	=	100 ares	= 1 hectare
0.386 square mile	=	1 square kilometer	= 100 hectares

ROW FEET/ACRE FOR CERTAIN DISTANCES:

Distance between rows (inches)	Feet of row/acre
12	43,560
18	29,010
24	21,758
30	17,427
36	14,526
42	12,439
48	10,853

DILUTION OF LIQUIDS AT VARIOUS CONCENTRATIONS

DRY MATERIALS (wettable powders):

Dry material required for—			
1 gal water	5 gal water	25 gal water	100 gal water
Tsp		*Oz*	*Lb*
1	3 tbsp	4	1
2	1½ oz	8	2
1	2½ oz	12	3
4	3¼ oz	16 (1 lb)	4
5	4 oz	20 (1¼ lb)	5

LIQUID MATERIALS (concentrates and so forth):

Liquid material required for—			
1 gal water	5 gal water	25 gal water	100 gal water
Tsp	*Fl oz*	*Oz*	*Pint*
½	½ (1 tbsp)	2	½
1	1	4	1
2	2	8	2
3	2½	12	3
4	3	16 (1 pint)	4

DILUTIONS:

	1:100	*1:200*	*1:400*	*1:800*	*1:1,000*
1 gal	2 tbsp + 2 tsp.	4 tsp	2 tsp	1 tsp	¾ tsp
3 gal	½ cup	¼ cup	2 tbsp	1 tbsp	2¼ tsp
5 gal	¾ cup + 5 tsp.	6½ tbsp.	3 tbsp	1 tbsp + 2 tsp.	1 tbsp + 1 tsp.
15 gal	1 cup + 3 tbsp.	½ cup + 2 tbsp.	4 tbsp + 2½ tsp.	3 tbsp + 2½ tsp.	1 pint + ½ cup.

PERCENT SOLUTIONS:

Percent	*Dilution*	*P/m*	*Grams/liter*
1.0	1:100	10,000	10.0
.1	1:1,000	1,000	1.0
.01	1:10,000	100	.1
.001	1:100,000	10	.01
.0001	1:1,000,000	1	.001

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